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HYPOCHROMIC ANAEMIA IN SOUTH AFRICA: A STUDY OF THE INCIDENCE IN THE TRANSVAAL 1955—56

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Is hypochromic anaemia uncommon among South African Bantu? Is it common among South African Whites? These questions prompted the study reported here. A survey of the cases of hypochromic anaemia diagnosed in one year in the haematology laboratory of this Institute was carried out.

PLAN OF STUDY

The laboratory at the time this study was undertaken was a regional one, drawing its material from many non-White hospitals on the Witwatersrand, and also from hospitals in the country districts from an extensive area of the Transvaal. Table I indicates the number of haematological investigations (morphological studies only) done during the period of survey, which was 1 year, from 7 September 1955 to 6 September 1956.

TABLE I. ANALYSIS OF BLOOD COUNTS DONE DURING THE PERIOD OF STUDY

	Whites	*Bantu*
Total blood morphology studies	42,684	11,974
Total of anaemias	6,428 (15%)	3,092 (26%)
Total of hypochromic anaemias	842	506
Percentage that hypochromic anaemias form of total morphological studies	2·0%	4·2%
Percentage that hypochromic anaemias form of total of anaemias	13·1%	16·4%

A copy of every haematological report issued by the Department is surveyed by the author for classification and record purposes. During the period of study every report indicating the presence of a hypochromic anaemia was noted and the following details recorded: Race, age, sex, haemoglobin value, and mean corpuscular haemoglobin concentration or degree of hypochromia. Only the first report on a case was recorded, repeat investigations being ignored. At the end of the period of survey the information recorded was transferred to punch cards for analysis.

Standard routine haematological methods were used in this survey. The haemoglobin was estimated as oxy-haemoglobin by a photo-electric colorimetric method. The packed-cell volume was estimated by the method of Wintrobe[1] (in preference to red-cell counts). The minimum investigation qualifying for inclusion in this report was the performance of a haemoglobin estimation with comment on the red-cell appearances on the stained film. These comments are made by medically qualified persons. For a diagnosis of hypochromic anaemia the report had to indicate the presence of an anaemia having regard to the age and sex of the patient and either (1) a mean corpuscular haemoglobin concentration below 31% or, if a packed cell volume had not been done, (2) an unequivocal statement of the presence of hypochromia of the red cells as shown by inspection of the stained smear.

RESULTS

Case Rate of Hypochromic Anaemia

Among 42,684 blood counts on Whites (this total includes repeat counts done during the period) 842 initial diagnoses of hypochromic anaemia were made. During the period 6,428 cases of anaemia of all types (i.e. cases with a haemoglobin value below the normal range) were encountered, so that 13·1% of all anaemias amongst Whites were hypochromic.

11,974 blood counts (including repeat counts) were done on Bantus, and 506 cases of hypochromic anaemia were encountered among 3,092 cases of anaemia of all kinds, i.e. 16·4%.

It is thus seen that the occurrence of hypochromic anaemia in the hospital populations of the races studied is very similar; at all events the occurrence rate among Bantu hospital patients is not less than among Whites.

Analysis by Place of Residence

Although the manner of life of the Whites differs little fundamentally as between town and country, much is made of differences between urban Bantu and rural Bantu. The data was therefore analysed according to residence; (1) within Johannesburg municipal area, (2) outside Johannesburg municipal area, and (3) a special category for mine Natives, who are almost entirely migrant labour. Table II sets out the data so analysed. Of the Whites, 74% were from within the Johannesburg municipal area; of Bantu 35% were from the municipal area, 44% were from outside and 21% were mine workers. Unfortunately, without knowledge of the composition of the population sampled, it is impossible to say from these figures whether hypochromic anaemia is commoner in the urban Bantu or in the 'rural' Bantu. It is, however, possible to compare the severity of the anaemias found in the two groups (see below).

TABLE II. ANALYSIS BY RESIDENCE

Origin	*Males*	%	*Females*	%	*Both Sexes*	%
			Whites			
Johannesburg Municipal area	240	80	384	71	624	74
Other..	61	20	157	29	218	26
Total ..	301	36	541	64	842	100
			Bantu			
Mines	107	—	—	—	107	21
Johannesburg Municipal area ..	69	39	111	41	180	35
Other..	107	49	112	51	219	44
Total ..	283	44	223	56	506	100

Age and Sex Analysis

(*a*) *Whites.* The sex ratio for Whites was M : F : : 1 : 1·8. Analysis according to age (Fig. 1) shows conspicuous differences in the distribution of the cases according to sex. There is a very high occurrence rate (almost 1/3rd of all the cases) in the under-10 age-group for males (Fig. 1). Table III sets out a more detailed analysis of the under-10 age-group. As would be expected, in females there is a high incidence in the 3rd and 4th decades with the peak incidence in the 5th decade; a sharp drop occurs in the 6th decade. In males, on the other hand, apart from the high incidence in childhood, there is a steadily increasing occurrence rate up to the 7th decade.

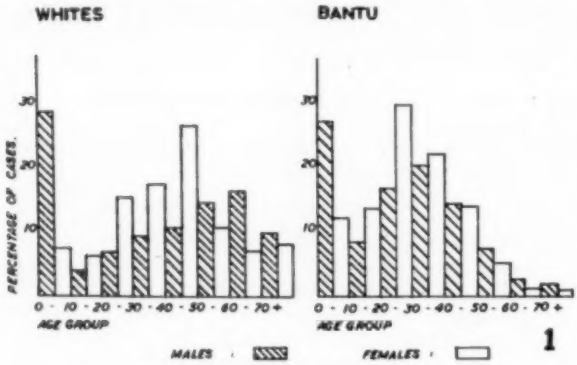

Fig. 1. Age and sex analysis by race.

(*b*) *Bantu.* The sex ratio of the civil population (i.e. excluding mine Natives) was M : F : : 1 : 1·27. When analysed by age the figures show the same higher incidence in boys than in girls in the under-10 age-group observed in the Whites (Fig. 1 and Table III). The life-expectancy curve for the Bantu is reflected in both the male and female hypochromic anaemia occurrence-rate curves. Thus, unlike the steadily climbing figures for White males, the incidence of hypochromic anaemia in the Bantu male reaches its peak (apart from the childhood one) in the 4th decade. Similarly, in Bantu women the incidence of hypochromic anaemia (probably due to gynaecological and obstetrical causes) reaches its peak in the 3rd decade. In both males and females the occurrence rate over the age of 50 is very small.

TABLE III. ANALYSIS OF UNDER-10 AGE-GROUP BY AGE AND SEX

Age	*Under 1*	*1 – 2*	*2 – 3*	*3 – 4*	*4 – 5*	*5 – 10*
			Whites			
Boys ..	27	19	3	0	0	4
Girls ..	6	11	2	6	1	1
			Bantu			
Boys ..	12	10	5	5	2	10
Girls ..	3	3	5	2	0	9

Severity of the Anaemia at Diagnosis in the Two Races

Table IV shows the severity of the anaemia present at initial diagnosis for the two races. The figures are very similar and do not suggest any great difference in the degree of anaemia necessary to make the individual, White or Bantu, seek medical care. The table may also suggest that the very many more blood counts done on Whites than on Bantu (3 times as many) represent so much time and money misspent; that is to say, that the apparently more elaborate care afforded to the Europeans failed to detect any more cases among them in the less severely anaemic categories.

TABLE IV. ANALYSIS OF SEVERITY OF ANAEMIA AT DIAGNOSIS

Haemoglobin Value at Diagnosis (GM%)	*Whites*		*Bantu*	
	No.	*%*	*No.*	*%*
Under 3	4	0·5	6	2·1
3·1 – 6	77	9·1	28	10·0
6·1 – 9	346	40·0	96	34·0
9·1 – 12	365	43·5	118	41·4
> 12	50	6·0	35	12·4

Severity of the Hypochromia present at Diagnosis

Table VA shows the percentage of cases classified according to the MCHC i.e. according to the severity of the iron deficiency. The MCHC value was available in 433 White cases (52%) and in 214 Bantu cases (42%). The figures might suggest that a higher proportion of the most severely hypochromic anaemias occur in the Whites, but the possibility of bias in selection of the cases on which the packed-cell volume was done throws doubt on the validity of this suggestion. In a comparison of urban Bantu and 'rural' Bantu and urban Whites and other Whites (Table VB) it was apparent that the most severely hypochromic cases were 'rural' in both cases. This possibly suggests that both urban Whites and urban Bantu seek medical attention earlier than their 'rural' counterparts.

TABLE VA. ANALYSIS OF DEGREE OF HYPOCHROMIA AT DIAGNOSIS

	MCHC <25·0% (Percentage)	*MCHC 25·0 – 27·9% (Percentage)*	*MCHC 28·0 – 31·0% (Percentage)*
White males ..	9·6	29·0	61·3
White females ..	9·0	31·2	59·7
Bantu males ..	7·0	19·0	73·0
Bantu females ..	2·5	31·3	66·1

TABLE VB. ANALYSIS OF DEGREE OF HYPOCHROMIA AT DIAGNOSIS BY PLACE OF RESIDENCE

	MCHC <25·0% (Percentage)	*MCHC 25·0 – 27·9% (Percentage)*	*MCHC 28·0 – 31·0% (Percentage)*
Urban White males	9·6	31·6	58·7
'Rural' White males	12·9	29·0	51·0
Urban White females	1·6	32·7	65·6
'Rural' White females	22·1	31·6	46·2
Urban Bantu males	3·2	21·7	75·0
'Rural' Bantu males	11·1	19·4	69·4
Bantu Mine workers	5·7	17·1	77·1
Urban Bantu females	—	23·1	76·8
'Rural' Bantu females	6·7	42·2	51·0

Analysis of Causation

This unfortunately was not possible in detail owing to the inadequacy of the information provided in the majority of cases by the clinician requesting the blood examination. However, among the 239 White males where a diagnosis was given, the commonest diagnosis were secondary haemorrhage (site unspecified) and haemorrhage from the gastro-intestinal tract, followed by infective conditions, mainly tuberculosis. This was followed closely by neoplastic conditions. No diagnosis of primary ('idiopathic') hypochromic anaemia was ventured. Diagnoses capable of classification were provided for 325 White female patients. Among these, secondary haemorrhage (site unspecified) was by far the commonest diagnosis given. Gynaecological bleeding was the next most frequent diagnosis but, interest-

ingly, it was equalled by gastro-intestinal haemorrhage. Anaemia associated with pregnancy was the next most frequent diagnosis given. 'Idiopathic' hypochromic anaemia was volunteered as a diagnosis in 5 cases.

In the Bantu a very different pattern is apparent. The first 4 most frequent diagnoses in 231 males were (1) infections (unspecified), (2) tuberculosis, (3) respiratory disease, and (4) gastro-intestinal haemorrhage. For 182 females the order was (1) infections, (2) tuberculosis, (3) gynaecological bleeding, (4) respiratory disease. It may be legitimate to wonder whether 'infections' and 'respiratory disease' are not synonyms for tuberculosis. If this were so, 43% of the Bantu female cases of hypochromic anaemia for whom diagnoses were given and 58% of the male cases may have been due to tuberculosis. The diagnosis of primary ('idiopathic') hypochromic anaemia was given once for a Bantu female patient, and twice for Bantu male patients. Of interest was the association of hypochromia with megaloblastic anaemia in 6 Bantu patients.

DISCUSSION

It is said that hypochromic anaemia is uncommon or even very rare among the Bantu.[2-5] No adequate data have ever been brought forward to support this statement. So far as I am aware no survey of the occurrence rate of hypochromic anaemia in South Africa, either in Bantu or European, hospital cases or others, has been published. This survey has shown that in both races hypochromic anaemia constitutes a similar proportion of all the anaemias as seen in hospital populations in the Southern Transvaal. The condition therefore cannot be said to be rare or uncommon, although the populations studied did not permit assessment of true incidence for either racial group.

The overwhelming majority of cases of hypochromic anaemia result from blood loss. Cases due solely to deficiency of dietary iron are excessively rare, if in fact they ever occur. Malabsorption syndromes, e.g. steatorrhoea, account for a small number of cases of hypochromic anaemia.

The question of the so-called 'idiopathic hypochromic anaemia' is a vexed one. The author's opinion is that if it occurs at all it is an exceedingly rare condition, whether in White or Bantu and whether in South Africa or in Europe. In the great majority of cases perfectly adequate explanations can be found for the iron deficiency without invoking that cloak for ignorance, 'idiopathic'. It is possible that a proportion of these cases are in fact cases of malabsorption syndrome.[6]

The possibility that some cases of hypochromic anaemia (other than thalassaemia) may be due to failure to utilize iron for haemoglobin synthesis in one on which there is little information. It is well known that conspicuous siderosis of many organs occurs in the Bantu.[7-10] Whether this iron is available for haemopoiesis and whether evidence of hypochromia of the red cells only appears after these iron 'stores' are exhausted are questions which await answers.

Infection and sepsis are known to cause defective utilization of iron for haemoglobin synthesis.[11,12] It would appear from this survey that infection may be a more important aetiological factor in the Bantu cases of hypochromic anaemia than in White cases.

The higher occurrence rate of hypochromic anaemia in boys, both White and Bantu, than in girls in the same age group, is of considerable interest and, apart from Woodruff,[13] who last year reported similar findings from the USA, no other reference has been found. It seems likely to be due to the different growth rates of the male and female infant in the 1st year of life. Drabkin[14] states that growth in haemoglobin in the 1st year is 40 GM for male infants and 30 GM for female infants. The rate of somatic growth is more rapid in boys than in girls. According to Jackson and Kelly,[15] boys on an average gain 16 lb. in their first year of life as compared with 13 lb. in girls. The demand for iron, therefore, is greatest during the first year of life and this period shows the maximum incidence of hypochromic anaemia in childhood (Table III).

SUMMARY

The occurrence rate of hypochromic anaemia in White and Bantu hospital populations in the Southern Transvaal has been compared. Hypochromic anaemia is shown to be by no means rare in the Bantu. Sex and age differences and differences in severity in the two races are analysed. Possible aetiological factors are considered.

The invaluable help of Miss J. Wilson in recording the data and preparing the punch cards for analysis is gratefully acknowledged.

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STUDIES OF AMINO ACID HANDLING IN KWASHIORKOR WITH A POSSIBLE EXPLANATION FOR THE INCREASED AMINO-ACIDURIA*

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Approximately 2 years ago we reported at Research Forum our observations on the increased amino-aciduria of 8 infants with kwashiorkor (protein malnutrition), who received both natural and synthetic meals. Since that time we have accumulated evidence† from an additional 8 cases confirming: (1) That the amino-aciduria reflects an increased excretion of 2-10 times the figure in our controls; (2) that it is the result of increases in at least 7 amino acids, and (3) that the increased excretion is temporary, returning to control levels after approximately 3 weeks of treatment. We are at present more particularly concerned with the possible mechanisms which may be responsible for this phenomenon.

Studies of renal function were designed in which 5 infants with kwashiorkor were fasted for 6 hours and were then given a test meal of skimmed milk providing approximately 1 g. of protein per kg. of body weight. Blood was taken every half hour and

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urine collected every 15 minutes by catheter for a 3-hour period following the test meal. Total amino acids were estimated in the urine and plasma by the Van Slyke gasometric technique. Urinary creatinine was also estimated.

It would appear that, in 4 of these cases, there was a tendency for the plasma amino acids to rise higher or remain at their maximal concentration for a longer time after the test meal; in fact, these increases are significantly greater ($p < 0{\cdot}02$) than those of the control series. This tendency is most pronounced in the severely ill cases and is reduced with treatment.

The same tendency also appears in the data on urinary amino acid, viz. the most severely ill infants show the greatest urinary excretion of amino acids, and this excretion is reduced on treatment.

More important for the purpose of this investigation, however, is the observation that in these cases of kwashiorkor, there appears to be a relationship between the concentration of plasma amino acid and the degree of amino-aciduria: wherever the plasma concentrations were highest, the urinary excretion of amino acids was greatest. It would appear, therefore, that renal function, in so far as amino-aciduria is concerned, is probably normal in kwashiorkor, but that the utilization of amino acids is altered, thereby causing the renal threshold to be exceeded. There is also some suggestion of a relationship between the urinary excretion of amino acids and creatinine—presumably a common, renal mechanism. The explanation that this is an artifact resulting from errors inherent in the 15-minute urine collection interval, appears justified by further investigations.

In an additional investigation 24-hour urine collections were made throughout the treatment of 4 cases of kwashiorkor and compared with collections made over a period of 2 weeks from a normal, active child of a similar age living in a home environment and eating a mixed diet.

It was observed that the urinary excretion of amino acids increases to a maximum within the first week of adequate treatment and is not related to fluctuations in creatinine excretion. These urinary amino acids were partitioned during 3 phases in treatment: on the first 2 days, on 2 days at maximal excretion, and on the final 2 days, at cure. These partitions confirm our earlier observations that the increased amino-aciduria of infants with kwashiorkor results from increases in the excretion of at least 7 amino acids.

SUMMARY

Renal function and other studies suggest that the increased amino-aciduria of kwashiorkor is due to altered utilization of absorbed amino acids causing the renal threshold to be exceeded. As yet, we have no evidence of a defect in renal tubular function.

PATCHY SENILE ELASTOMA OF THE LIP RESEMBLING LEUKOPLAKIA

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In 1955 I was asked to see a 49-year-old spinster who was obsessed with an opalescent spot which she had on the external vermilion of the lower lip. There was no atrophy or epithelial change, and the nature of this solitary focal lesion was not clear to me. Histologically it consisted of an area of senile elastosis without any epidermal or other alteration (Fig. 1). It was interesting to note that whitening of the lip could result from elastosis alone, that the lesion could be a papular 'elastoma', and that no epithelial change or leukoplakia need occur with it.

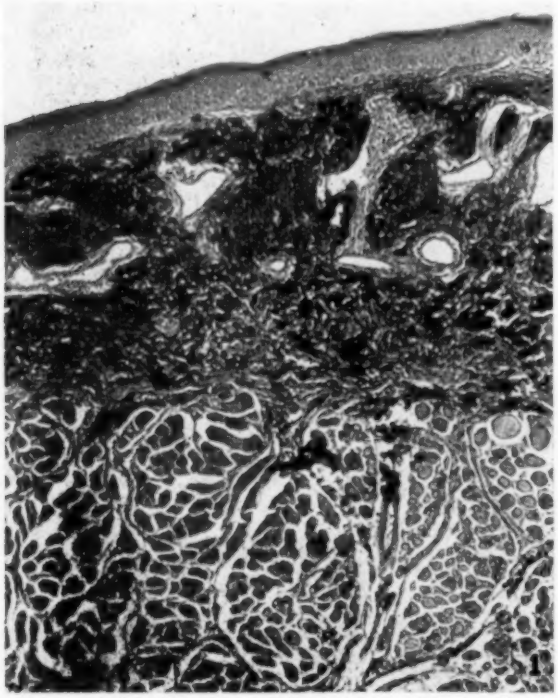

Fig. 1 Section of lower lip showing elastoma between the epidermis and muscle. Weigert's elastic stain.

Since then I have seen the same thing repeatedly, and also observed nodular senile elastomas of the cheek suggesting neoplasm. As a precancerous manifestation, lip elastosis is associated with loss of substance, chronic inflammation and a readily damaged horny layer, thick, thin or scaly.[1] Whitening or leukoplakia is then epithelial. Whitening from dermal change alone is nevertheless seen at times in lichen sclerosus, though whitening on the lip or vulva usually comes from a thick and sodden horny layer, a thick Malpighian layer, or both. Whitening from an increased granular layer is a poorly substantiated possibility. Senile elastosis on the open skin tends to look yellow, perhaps from the liberation of fluorescent pigments[2] or lipids, though in my experience lipids are colourless. Perhaps the incident-light microscope may contribute in the study of these dermal colour changes.[3]

Elastic tissue and elastomas have interested a number of workers in this country, and it may be of value to note at least a few of the contributions. Pepler and Brandt[4] and the present writer[5] have investigated the staining and enzymatic dissolution of elasti tissue. Sacks[6] has also reported work on the same topic. Although the derivation of senile elastosis from elastic tissue was held by myself and recently supported by Braun-Falco in Germany,[7] Gillman in Durban has taken the view that its parent substance is collagen.[8,9] Marshall and Lurie[10] have contributed to our knowledge of another kind of focal elastoma, while Loewenthal[11] has studied the changes in certain rare varieties of connective-tissue naevus.

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VAN DIE REDAKSIE : EDITORIAL

SNYKUNDIGE BEHANDELING VAN MISVORMINGE VAN DIE ANUS EN REKTUM BY KINDERS

Soos dit blyk uit die oorsigtelike artikel van prof. J. H. Louw wat ons op bladsy 874 van hierdie uitgawe plaas, het die snykunde van misvorminge van die anus en rektum by kinders geweldige groot vooruitgang gemaak gedurende die afgelope aantal jare. Trouens, alhoewel die werk wat op hierdie gebied gedoen word nie met sulke skouspelagtige vertoon ontvang is as werk op ander gebiede van die medisyne nie, is die betekenis en implikasies daarvan in terme van die kwaliteit van die werk wat gedoen word, sowel as van die mate van menslike gesondheid en geluk wat daardeur bevorder word, net so ongeëwenaard.

In weerwil van die vooruitgang op hierdie gebied, en juis ook omrede daarvan, bly daar egter tog nog 'n aantal belangrike oorwegings wat in hierdie verband benadruk moet word. Om mee te begin moet ons aantoon dat, alhoewel dit nog steeds die belangrike taak van die algemene praktisyn bly om die toestande waaroor ons skryf vroeg raak te sien en te diagnoseer, dit eweneens die taak van 'n deskundige met spesiale opleiding en ervaring bly om die meeste van die toestande te behandel.

Tot groot nadeel en skade van die pasiëntjies en van hulle ouers word snykundige behandeling van kongenitale misvorminge van die anus en rektum by kinders nog te dikwels aangedurf deur welmenende dokters wat egter nie volledig genoeg vir die spesifieke taak toegerus is nie omdat hulle nie oor gedetailleerde besonderhede van die tegniek beskik nie, en ook omdat hulle nie bekend is met die langtermyn uitslae van snykundige werk op hierdie gebied nie. Die onlangse waarskuwing van Potts[1] is dus hier werklik ter sake: ‚Is dit reg dat 'n chirurg wat nie daarvan sou droom om te opereer vir 'n tumor van die brein of 'n gesplete verhemelte nie, sal kans sien om 'n operasie uit te voer waar daar atresie van die rektum is by 'n seuntjie met 'n rektovesikale fistel—sonder dat die dokter ooit so 'n operasie sien doen het? . . . Sulke gevalle . . . word gewoonlik swak gehanteer en die pasiënte is dikwels gedoem tot blywende inkontinensie of 'n permanente kolostomie.'

Om die probleme in hierdie verband in die regte perspektief te stel, wys professor Louw op die mees opvallende voorbeelde van wanopvattings en mistastinge wat daar nog steeds op hierdie gebied gemaak word. In die eerste plaas is daar byvoorbeeld die opvatting dat geslote anus gewoonlik 'n anomalie is waarby net 'n dun membraan die weg van die mekonium versper. In werklikheid kom hierdie soort membraanversperring slegs by 'n klein persentasie van gevalle voor. In die 85 gevalle wat hy behandel het, het professor Louw slegs eenkeer hierdie diagnose gemaak.

Tweedens moet daar gewys word op die gevaarlike praktyk om 'n poging te maak om die gaping tussen anus en kolon te oorbrug deur middel van 'n perineale benadering. Hierdie soort operasie word dikwels uitgevoer by ongeskikte gevalle, met die gevolg dat die kind vir die res van sy lewe met 'n ernstige rektale swakheid bly sit.

Derdens is daar nog altyd die goedgelowige aanvaring van die opvatting van fistels tussen die rektale sak en die urineweë, die vagina, of die vel. Hierdie opvatting is gebaseer op 'n verkeerde interpretasie van die embriologiese ontwikkeling, en dit lei tot onbevredigende behandeling.

Vierdens word die fout begaan om 'n inguinale kolostomie aan te raai in die gevalle van pasiënte waar 'n kolostomie nodig skyn te wees. Deur dit te doen word die oortollige lis van die sigmoïede kolon, wat anders vry sou wees om gebruik te word by rekonstruktiewe chirurgie in die toekoms, opgeoffer. Indien 'n kolostomie nodig is, moet dit in die dwars kolon geplaas word om soveel moontlik van die distale deel van die kolon te laat vir gebruik by 'n latere operasie.

Die feit dat professor Louw in die artikel waarna ons verwys 'n deurtastende ontleding gee van die anatomiese, embriologiese, kliniese, radiologiese en terapeutiese aspekte van die belangrikste probleme in hierdie verband, moet dus as 'n besondere bydrae beskou word. Werk van hierdie aard, alhoewel dit sy eie besondere tegniese fasette het, is dus nie net vir die deskundige chirurg op hierdie gebied van belang nie, maar ook vir die groot aantal praktisyns wat in hulle daaglikse werk voor hierdie soort probleme te staan kom.

1. Potts, W. J. (1958): J. Amer. Med. Assoc., 166, 462.

CYSTS OF THE LUNG IN CHILDREN

Pulmonary cysts comprise an interesting group of abnormalities, over which there is still controversy. A cyst may be defined as a pathological space-containing fluid, or semifluid, or air, and lined by epithelium or endothelium.[1] Broadly speaking cysts may be classified into two groups, congenital and acquired. Acquired cysts may develop through a variety of mechanisms, viz. check-valve bronchial obstruction, pulmonary necrosis (bacterial and fungal), echinococcal infection, and trauma. These cysts, on the whole, present fewer problems than do the congenital cysts and will not be discussed further.

It has long been believed that lung cysts are of developmental origin. Such arguments as the frequent finding of cysts in infants and children, and the occasional co-existence of true developmental anomalies (for example, the association of cystic bronchiectasis with transposition of the viscera in Kartagener's syndrome), have been used to support this concept. Moersch and Clagett[2] divided these cysts into two groups: those of bronchial origin and those arising from alveoli. Bronchogenic cysts possess a lining of columnar or cuboidal cells, frequently have cartilaginous or muscle elements in their outer wall, are often filled with fluid, and are situated adjacent to the lung root. Cysts of alveolar origin differ in that the lining membrane is of alveolar cells; the cyst

is invariably filled with air and is more peripherally situated. Caffey[3] has produced evidence against these cysts being of congenital origin, and he believes that most, if not all, of the so-called 'congenital cysts' may be explained on the basis of check-valve bronchial obstruction. The bronchial elements in the cyst wall can be reasonably explained by the purely mechanical factors of bronchial dilatation beyond bronchial obstruction.

Perhaps the most reasonable view is that of Campbell,[4] who holds that developmental bronchopulmonary malformations, through a variety of check-valve malformations, may give rise to lung cysts. Embryonic blockage of the lumina of the bronchial buds occurs, and single or multiple cystic cavities, lined with bronchial structures and filled with secretions, results. This hypothesis would also explain the finding of cysts in sequestration of the lung.

This controversy has its practical applications, for the distinction as to whether a lesion is of congenital or acquired origin may be vital to prognosis and treatment. A congenital anomaly is more likely to be treated surgically on the basis that it is irreversible, while an acquired lesion is more apt to regress spontaneously.[4] In following up a series of thirteen infants with lung cysts over a number of years, Caffey found that regression occurred in all. Complications of lung cysts include the development of infection, tension, and spontaneous pneumothorax; but it seems that a conservative non-operative approach should be followed until the irreversible nature of the condition is firmly established, or critical respiratory distress makes surgical intervention urgent.[4]

1. Lodge, T. (1952): Proc. Roy. Soc. Med., **45**, 629.
2. Moersch, H. J. and Clagett, O. T. (1947): J. Thorac. Surg., **16**, 179.
3. Caffey, J. (1953): Pediatrics, **11**, 48.
4. Campbell, J. A. (1955): Amer. J. Surg., **89**, 1009.

MALFORMATIONS OF THE ANUS AND RECTUM*

A REPORT ON 85 CONSECUTIVE CASES

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'Imperforate anus' is a congenital anomaly which is commonly encountered in practice. Indeed, it is the commonest of the congenital malformations that are incompatible with life.[1] The incidence of this anomaly is about 1 in 5,000 live births[2] and we have had the privilege of treating no less than 85 cases during the past 6 years.

It should be clear that a large number of surgeons encounter the lesion during their career and that operations on babies with this condition are often performed by men who are unfamiliar with the technique and results of surgery in these cases.[1] In this connection I should like to quote Willis Potts[3] who recently stated: 'People are biased (most people are) but is it quite fair that a surgeon who would not think of operating on a brain tumour or repairing a cleft palate should tackle a case of atresia of the rectum in a male infant with a rectovesical fistula when he never before has seen such a case nor witnessed the operative repair? . . . Such cases . . . are apt to be poorly handled. If the defects are not properly corrected at the first operation, these patients may be forever incontinent or doomed to a permanent colostomy.'

The following are examples of some of the mistakes that are still being made in the diagnosis and management of imperforate anus:

1. The popular idea that 'imperforate anus' *in its common form* is an anomaly where only a thin membrane prevents the escape of meconium (Fig. 1) 'which bulges the membrane *through the anal canal* as a plum-coloured mass'.[4] Although Gross[2] found the anomaly in 2·8% of his cases of anorectal malformations, it is generally agreed that this kind of membrane is excessively rare,[5] and Denis Browne,[6] in his wide experience, has never encountered a case. We have made the diagnosis on one occasion in our series of 85 cases, but the 'membrane' was no longer intact and we assumed that it had been perforated by the person who had originally attended the infant. It should therefore be clear that in the treatment of 'imperforate anus' the advice that 'all that is necessary is to incise the septum crucially'[7] is seldom applicable.

The commonly accepted classification of malformations of the anus and rectum is that of Ladd and Gross[8] in which 4 main visceral types, with and without 'fistulae' are recognized (Fig. 2). This classification, however, is based on an erroneous interpretation of the embryology (*vide infra*), and fails to act as a guide to treatment and prognosis. We have adopted a modification of Denis Browne's[9] classification elaborated by Douglas Stephens[10,17] and based on the work of Wood Jones[11]

TABLE I. ANORECTAL MALFORMATIONS, 85 CASES

Rectal agenesis, 32 cases	
1. Recto-urethral fistula (M)	27
2. Recto-vesical fistula (M)	1
3. Recto-vaginal fistula (F)	2
4. Without fistula (F, M)	2
Anal anomalies, 47 cases	
1. Ectopic anus (26)	
(*a*) Perineal (M)	1
(*b*) Perineal (F) 'Shot-gun anus'	2
(*c*) Vestibular (F)	16
(*d*) Vaginal (F)	7
2. Covered anus (18)	
(*a*) Ano-perineal 'fistula' (M)	15
(*b*) Ano-bulbar 'fistula' (M)	1
(*c*) Ano-vulvar 'fistula' (F)	2
3. Imperforate anus (M)	2
4. Imperforate anal membrane (M)	1
Gross malformations, 6 cases	

M = Male F = Female

and Sir Arthur Keith.[12] In this scheme 2 broad groups of abnormalities are recognized, viz. rectal and anal (Table I).

2. The common and most dangerous practice of attempting to bridge the gap between anus and colon by a perineal approach.[1] This is only too frequently done in unsuitable cases at the expense of muscles, nerve supply and blood supply, and the child is left a rectal cripple. A popular textbook of surgery[7] advises perineal dissection when 'the distance

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between the end of the rectum and anal dimple is not more than 2 inches (5 cm.)', but most enlightened authorities on the subject agree that such dissection is highly dangerous when the distance exceeds 1·5 cm.[1,2,13-16] Even the arbitrary figure of 1·5 cm. is not a satisfactory criterion, [10,17] and the wise surgeon will depend on anatomical landmarks rather than a ruler in coming to a decision.

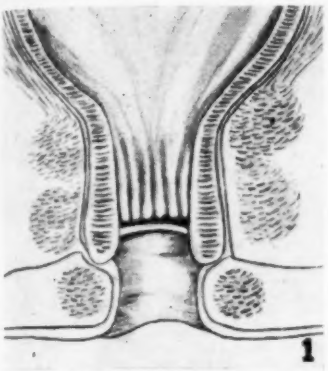

Fig. 1. Diagram of imperforate anal membrane often referred to as the 'common form' of 'imperforate anus'. In practice it is excessively rare.

3. The almost blind acceptance of the concept of 'fistulae' between the rectal pouch and urinary tract, vagina or skin.[2,15,16] Such 'fistulae' occur in more than 70% of cases[2] and while this idea of a 'fistula' prevails, treatment by 'transplantation of the fistula' will continue, and must remain unsatisfactory. This idea is based on an erroneous interpretation of the embryology which is still tenaciously accepted by many people despite Wood Jones'[11] observation more than 50 years ago that 'the generally accepted view of the development of the human hind end offers no explanation of some of the abnormalities which are commonly found clinically in this situation', and Sir Arthur Keith's[12] finding that 'In the series of malformations now described to you (114) there is not one single form that is satisfactorily accounted for thereby'. Most 'fistulae' should be treated by much simpler and less destructive procedures than 'transplantation'.

4. The mistake is made of advising inguinal colostomy in patients where a colostomy seems necessary.[7] In doing this, the redundant sigmoid loop, which should be left free for future reconstructive surgery, is sacrificed. A colostomy, if required, must be placed in the transverse colon to leave as much distal colon as possible for use at the definitive operation.[1,9]

ANALYSIS OF MATERIAL

Our cases of 'imperforate anus' have been divided into the 2 main groups, viz. rectal and anal (Table I). There were 32 cases of rectal agenesis and 47 with anal malformations. (A third group consisting of 6 cases and designated 'gross malformations' has been included for the sake of completeness.) This classification has enabled us to determine the necessary form of treatment and to estimate with reasonable accuracy the prognosis in each individual case. The 2 groups differ in many respects, viz. anatomically, embryologically, clinically, radiologically, as well as therapeutically and prognostically. These differences will be discussed briefly. (In the group with gross malformations the whole 'hind end' was deformed with multiple congenital anomalies and extroversion of the cloaca. These malformations are not amenable to surgery and will not be discussed.)

A. Anatomy

The most important difference between rectal and anal anomalies is to be found in their relationship to the levator ani muscle.[10,17] In rectal anomalies the bowel ends blindly above the levator whereas in anal anomalies it extends through the levator and is embraced by the puborectalis sling. It should be noted that in the newborn, the level of the levator is just below a line drawn from the symphysis pubis to the sacrococcygeal junction (P–C line, Fig. 3). In the normal infant this line marks the positions of verumontanum in males, the external os of the cervix in females, the peritoneal reflection, and the third fold of Houston.[10,17,18]

Rectal Agenesis, 32 *Cases*

Boys are much more frequently affected than girls (29 of our cases, Table I). In the common type in males the bowel ends on the pubococcygeal line and communicates *via* a minute 'fistula' into the prostatic urethra (Fig. 4a). The bowel rarely terminates in the bladder (1 of our cases). The anal canal is often absent and the external anal musculature in this region is usually entirely vestigial.[10,17,19] The levator ani itself may be poorly developed particularly in cases associated with absence of more than 2 sacral vertebrae.[10,14,17,20] In females the fistula opens high up into the posterior fornix of the vagina (Fig. 4b). A very rare variant in both sexes is agenesis without a 'fistula'.

Anal Anomalies, 47 *Cases*

Two common and 2 rare varieties can be recognized (Table I):

(*a*) *Ectopic anus*, 26 *cases*. Girls are much more commonly affected than boys (25 of our cases, Table I). The rectum passes through the levator which forms an adequate sling around it but, instead of opening on the surface at the proctodeum, it passes obliquely forward and in females opens through a somewhat stenosed orifice as follows:

(i) On the perineum—'shotgun anus' with near-normal external anal muscles (Fig. 5a, 2 cases). The normal strip of skin separating vaginal and anal orifices is absent.
(ii) Into the vestibule—'vestibular anus' with poorly developed external anal muscles (Fig. 5b, 15 cases).
(iii) Into the lower third of the vagina—'vaginal anus' with vestigal external anal muscles (Fig. 5c, 7 cases).

In males the anus is simply placed more anteriorly than normal (Fig. 5d, 1 case), thus close to the posterior border of the scrotum. The external anal musculature is practically normal.

The proctodeal pit is obliterated in all these cases and the external anal musculature deficient in most. At best, the deep portion of the external sphincter is well-developed.

(*b*) *Covered anus*, 18 *cases*. Boys are most commonly affected (Fig. 6a, 16 cases). In them the perineal raphe extends posteriorly over the anal site where it is usually thickened to form a ridge or tag. The rectum terminates just deep to this tag opposite the bulb of the urethra.[10,17] The anal orifice is covered by skin and is often projected forwards along the median raphe to the perineum, scrotum, and penis, or even as far as the frenulum. The deep external sphincter is well developed, but the superficial part is represented by a thin collection of fibres passing in a sagittal plane on either side of the 'anoperineal fistula'.[10,17] A rare variant in boys (1 of our cases) is the so-called 'anobulbar fistula'.[10,17] The perineum is absent, the lumen of the anus is projected forwards into the bulb of the urethra, and hypospadias usually coexists.

In girls (Fig. 6b, 2 cases) the anus is similarly covered by skin and projected anteriorly parallel to the perineal plane

ADD AND GROSS CLASSIFICATION2
ECTOPIC ANUS

to open at, or close to, the fourchette in the fossa navicularis—'anovulvar fistula'. The orifice is constricted and the patients usually have some male characteristics, e.g. enlarged clitoris.[21] In very rare, exaggerated examples of this condition the vestibule is also covered and the anus projected forwards, together with the vagina to the base of the clitoris.[6]

(*c*) *Imperforate anus* (*Fig.* 7, 2 *cases*). This condition occurs almost exclusively in males. The rectum terminates blindly at the *upper* border of the bulbocavernosus,[10,17] and the anal dimple is absent or rudimentary. The external sphincters are poorly developed or rudimentary and incorporated with the bulbocavernosus.[10,17]

(*d*) *Imperforate anal membrane* (*Fig.* 8, 1 *case*). A thin membrane stretches across the lumen of the anorectal canal at the site of the anal valves, i.e. at the level of the triangular ligament or lower level of the bulbocavernosus.[10,17] All the muscles are intact except the superficial external sphincter which is rudimentary or absent.[22]

B. Embryology

Broadly speaking, it may be said that the rectum is derived from the entodermal cloaca and the anal canal from the ectodermal claoca (Fig. 26). It must be understood, however, that there is coordination in the differentiation of these two primitive structures, and that errors in the development of one may be accompanied by errors in the development of the other. More details cannot be discussed here.

The development of the *rectum* is intimately associated with that of the urogenital sinus—bladder, urethra, and internal generative organs (Fig. 9). At first there is free communication between the two structures, but this soon closes off due to downgrowth of the urorectal septum and ingrowth of the lateral folds of Rathke—Fig. 9.[23-25] Then the rectum has to 'find' a new 'vent' by migrating to the perineum and establishing a communication with the proctodeum or anal canal.[5,12,26]

In 'rectal agenesis' the development of the rectum is usually arrested at an early stage before it has closed off from the urogenital sinus.[26] The primary error is probably failure of fusion between the fused Rathke's folds below and the urorectal septum above.[25] Since this provides the bowel with a vent, migration is arrested and the rectum terminates in this region, i.e. the prostatic urethra. Concommitant arrest of development of the anal canal is common.[10,17]

The development of the *anal canal* is associated with the development of the pars phallica of the urogenital sinus (penile urethra in males and lower third of vagina and vestibule in females) and with the formation of the genital folds and anal tubercles (Fig. 9).[9,21,22,27] The usual primary error is either imperfect development or premature fusion of the anal tubercles, or excessive posterior fusion of the genital folds with obliteration of the 'proctodeal pit'.[10,21,22]

(*a*) *Ectopic Anus*

The anal tubercles and inner genital folds do not fuse to form a perineum and the rectum then fails to migrate posteriorly.[5,22] The ectopic anus may open more anteriorly on the perineum, but more frequently there is 'compensatory' incomplete fusion of the lateral folds of Rathke and the establishment of a communication between the terminal rectum and pars phallica of the urogenital sinus (vestibule and lower third vagina in females). The condition is more common in females because posterior fusion of the genital folds is normally defective.[22]

(*b*) *Covered Anus*

There is excessive posterior fusion of the labioscrotal folds and also of the posterior portions of the superficial part of the anal tubercles.[9,21] The anterior ends of the anal tubercles, however, do not fuse;[22] this occurs at a time when the rectum

Fig. 2. Ladd and Gross[8] classification of anorectal anomalies: 1. Stenosis. 2. Persistent membrane. 3. Blind rectal pouch. 4. Blind rectal pouch with normal anus. 'Fistulae' may occur in association with any of these.
Fig. 3. Diagram of normal anatomy: (*a*) Male, and (*b*) female. Note the relationship of the levator ani muscle and pelvic viscera to the pubococcygeal line (P–C).
Fig. 4. Diagram of rectal agenesis. The rectum terminates on the pubococcygeal line proximal to the levator ani. (*a*) Male with recto-urethral 'fistula', and (*b*) female with rectovaginal 'fistula'.
Fig. 5. Diagram of ectopic anus: (*a*) 'Shot-gun perineum' in female, (*b*) vestibular anus in female, (*c*) vaginal anus in female, and (*d*) perineal anus in male.
Fig. 6. Diagram of covered anus: (*a*) Male, 'ano-perineal fistula', and (*b*) female, 'anovulvar fistula' or vulvar anus.
Fig. 7. Diagram of imperforate anus.
Fig. 8. Diagram of imperforate anal membrane.
Fig. 9. 'Ectopic vaginal anus' with chronic constipation and overflow incontinence resulting in excoriation of vulva and perineum.
Fig. 10. Rectal agenesis. 'Blank' perineum with raphe crossing the normal site of the anus. Note the small buttocks and absence of natal cleft.
Fig. 11. Rectal agenesis. Urine containing meconium has left a distinct stain on the diaper.
Fig. 12. 'Shot-gun perineum'. Anal and vaginal orifices open side by side without an intervening skin bridge.
Fig. 13. 'Vestibular anus'. A stenosed anal orifice is situated in the vestibule. A probe introduced into this orifice passed in a cranial direction.
Fig. 14. 'Covered anus'. The anus is covered by a triangular skin tag and immediately in front of this meconium can be seen shining through the perineal skin. Examination of this area through a lens revealed a minute 'microscopic anus' with escape of gas and a 'fly-speck' of meconium.
Fig. 15. Covered anus. The anus is covered by skin only and a track of meconium passes forwards along the midline raphe, (*a*) just deep to the skin to burst through on the scrotum, or (*b*) on the ventral surface of the penis.
Fig. 16. Vulvar anus. The anus is covered by skin and meconium tracks forwards just deep to the skin to open in the fossa navicularis at the fourchette. A probe introduced into the opening passed dorsally, parallel to the perineal skin.
Fig. 17. Imperforate anus. The anal dimple is absent and filled in by a ridge of tissue. No meconium can be seen shining through.
Fig. 18. Diagram illustrating the principle of the 'cut-back' operation in vulvar, vestibular and low vaginal anus.
Fig. 19. Photograph of method of 'uncovering' a covered anus (Fig. 15).
Fig. 20. Anal stricture in a case of rectal agenesis treated by abdomino-anal reconstructive surgery.
Fig. 21. Excoriation of buttocks due to incontinence following abdomino-anal reconstruction of rectal agenesis.
Fig. 22. Result of 'cut-back' procedure for vestibular anus. The anomaly has been converted into the 'shot-gun' variety. (*a*) After 3 weeks, and (*b*) after 3 months.
Fig. 23. Result of 'uncovering' a covered anus; (*a*) After 1 week, and (*b*) after 3 months.
Fig. 24. Result of perineal anoplasty for imperforate anus. The anus has healed by granulation and tends to gape as indicated or become stenosed.
Fig. 25. Result of 'cut-back' procedure in vulvar anus. The posterior extension of the labial folds can still be seen. Continence is satisfactory and normal childbirth possible.

has already migrated, hence meconium collects under tension just deep to the perineal raphe and in due course finds its way forwards in the loose connective tissue just deep to the raphe, bursting through to the surface at any point between the proctodeum and tip of the penis. (In the anovulvar variety there is also failure of fusion of the inner genital folds.)

(*c*) *Imperforate Anus*

There is imperfect development and premature fusion in the midline of the anal tubercles, but the migrating rectum finds no external 'vent'.[22]

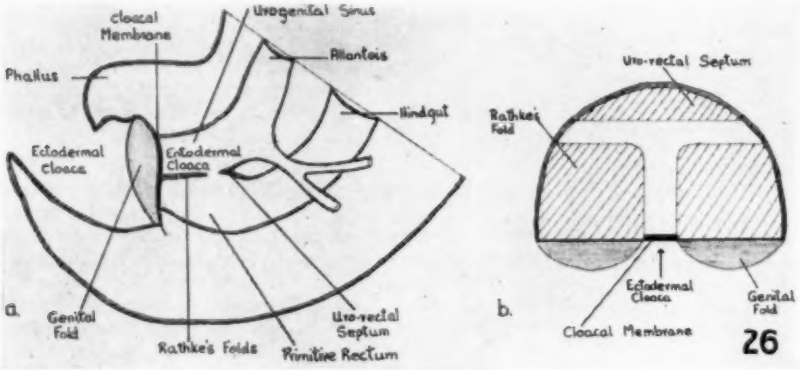

Fig. 26. Diagrammatic representation of human cloaca at 12-14 mm. stage. (*a*) Sagittal section, and (*b*) coronal section. Note the downgrowth of the urorectal septum and ingrowth of Rathke's folds which divide the cloaca into urogenital sinus ventrally and rectum dorsally. At the same time the genital folds and anal tubercles develop into the external genitalia and anal canal.

(*d*) *Imperforate Anal Membrane*

This is due merely to failure of breakdown of the proctodeal membrane or anal plate.[10,17]

C. Clinical Features

1. *Intestinal Obstruction*

The most striking and important clinical difference between rectal and anal anomalies concerns the development of intestinal obstruction.

In most cases of *rectal agenesis* symptoms and signs of acute intestinal obstruction become manifest within 48 hours. This happens in all cases without a fistula and also in boys with the common anomaly, viz. recto-urethral 'fistula', because the 'vent' is only minute. In the rare cases of recto-vesical fistula and rectovaginal fistula the opening may be large enough to deflate the bowel.

In most *anal anomalies* acute intestinal obstruction does *not* develop. In all varieties of ectopic anus the orifice is large enough to act as an efficient 'vent' during infancy. Later in life, however, when the stools become more solid, these children are prone to chronic constipation with faecal impaction, secondary megacolon, and overflow incontinence (Fig. 9). In patients with covered anus the thin skin which occludes the anal orifice usually ruptures spontaneously, and the same probably applies to that rare anomaly, imperforate anal membrane. Only in cases with imperforate anus and imperforate anal membrane does the patient develop acute intestinal obstruction, and differentiation from rectal agenesis must depend upon other features.

2. *Appearance of Perineum*

Rectal agenesis. In most of the cases the perineum is 'blank', often with a longitudinal median ridge running from the site of the normal anal position to join the median raphe of the scrotum (Fig. 10). Stimulation of this tissue may cause puckering due to contraction of the underlying vestigial muscle. The perineum may bulge when the baby cries, but meconium cannot be seen shining through the skin. There may be a dimple instead of a ridge and in rare cases a normal anal canal (Ladd and Gross, type 4) may be present. In many cases the buttocks are unusually small and the natal cleft obliterated (Fig. 10).[14]

In boys suffering from the common variant (recto-urethral fistula) there may be evidence of a communication with the urinary tract, e.g. 'the sign of green urine',[14] emergence of gas or a minute speck of meconium from the urethral meatus, flecks of meconium on the diaper (Fig. 11), or meconium in a centrifuged specimen of the urine. In females meconium may discharge from the vulva and, on passing a speculum, the fistula may be seen opening into the posterior fornix of the vagina.

Anal anomalies. The appearances depend on the type of anomaly:

(*a*) Ectopic anus. The anal orifice, which is somewhat stenosed but otherwise normal in appearance, is situated anterior to the normal position, e.g.:

(i) In the perineum both in males and females. In the latter case Denis Browne refers to the anomaly as 'shot-gun perineum' (Fig. 12).
(ii) In the vestibule (Fig. 13). A probe introduced into the orifice passes obliquely upwards, but can be felt through the perineum.
(iii) In the lower third of the vagina. A probe inserted into the orifice passes cranially, and only with forceful depression can it be felt through the perineum.

(*b*) Covered anus. In boys there is an epithelial fold suggestive of a hypertrophied perineal raphe extending over the usual anal site where it is often thickened to form a triangular posterior 'tag' (Fig. 14). Close scrutiny of what appears to be an intact perineum will reveal a bluish tinge shining through the skin and a small spot of meconium, no larger than a fly-speck, emerging from a microscopic opening adjacent to the 'tag'—the microscopic anus of Denis Browne (Fig. 14). In other cases a bluish track filled with meconium can be seen extending forwards (Figs. 15a and b). It usually bears little epithelial pearls and opens at the root of the scrotum or on the ventral surface of the penis. (In the anobulbar variety the anomaly is associated with a cleft scrotum and atypical hypospadias.)

In the vulvar anus of girls the anal dimple is also absent and filled by a central sagittal raphe. A track runs forwards and opens into the vulva close to the fourchette in the fossa navicularis (Fig. 16). A probe introduced into this opening passes directly backwards under the perineal skin. The

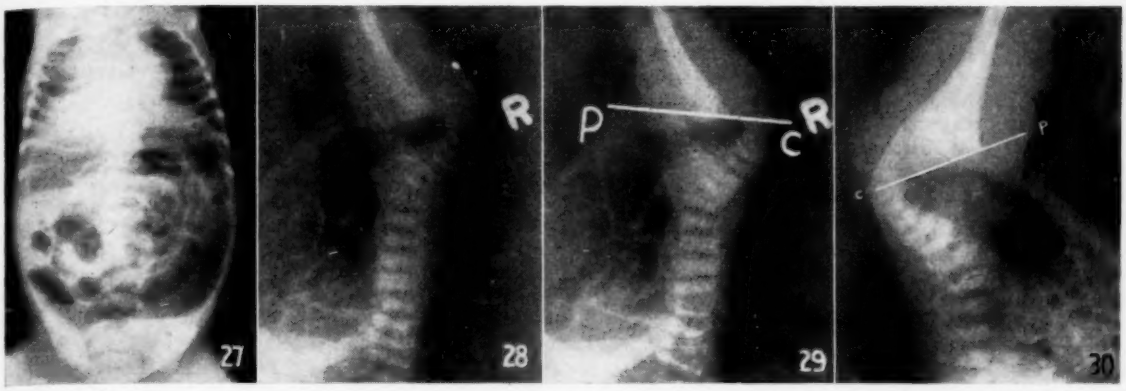

Fig. 27. Rectal agenesis. Flat A.P. plate (child erect) showing distended small and large bowel loops and fluid levels due to intestinal obstruction. (A similar picture is found in cases with imperforate anus.)

Fig. 28. Rectal agenesis. Flat, lateral plate (child inverted with marker at normal site of anus). The distance between the terminal gas shadow and anus is greater than 1·5 cm.

Fig. 29. Rectal agenesis. Flat, lateral plate (child inverted and pubococcygeal line inserted). The terminal gas shadow ends at the P–C line.

Fig. 30. Imperforate anus. Flat, lateral plate (child inverted and pubococcygeal line inserted). The terminal gas shadow extends caudal to the P–C line.

clitoris is often enlarged and resembles severe hypospadias so that in extreme cases of so-called 'covered vagina' the external genitalia resemble those of a boy.[6]

(*c*) Imperforate anus (Fig. 17). The appearance of the perineum is similar to that in rectal agenesis with a median perineal ridge, but there is *no* evidence of a recto-urinary fistula.

(*d*) Imperforate anal membrane. It is said that a normal anal canal is present and that the membrane bulges through it as a plum-coloured mass which becomes more obvious when the child cries.[4] When the membrane has ruptured the area may be stenotic with ragged epithelial tags at the level of the anal valves.

3. *Associated Anomalies*

Here, again, there is a striking difference between rectal and anal malformations (Table II).

TABLE II. SERIOUS ANOMALIES ASSOCIATED WITH ANORECTAL MALFORMATIONS

Type of anorectal malformations	*Anomalies of sacrum and coccyx*	*Anomalies of genito-urinary system*	*Other malformations*
Anal (47) ..	3	5	7
Rectal (32) ..	16	21	23
Gross (6) ..	6	6	6
Total (85) ..	25	32	36

In rectal agenesis there are frequently other abnormalities. Among these, vertebral anomalies are the commonest (50% of our cases), particularly the absence of sacral vertebrae.[10,14,17,20] These have an important bearing on the development of the levator muscles which are incompletely developed when more than two segments of the sacrum are missing. Urinary anomalies (70% of our cases) are very common[10,14,17] and malformations of the heart, oesophagus, intestinal tract, and nervous system may also occur.[2,28] In females associated malformations of the uterus and vagina are common.[10,17]

Anal anomalies are seldom associated with other serious anomalies (Table II), although vertebral malformations[20] and deformities of the external genitalia do occur.

D. Radiological Features

In patients who develop acute intestinal obstruction (mostly rectal agenesis; rarely imperforate anus) a straight X-ray of the abdomen will reveal distended small and large bowel with fluid levels (Fig. 27). This has to be differentiated from neonatal Hirschsprung's disease, the meconium-plug syndrome, and peritonitis with ileus. In cases with an adequate 'vent' the plates will be normal.

The position of the blind rectal pouch may be determined by holding the infant upside down and taking lateral films of the abdomen and pelvis. Gas in the bowel rises to the top, displacing meconium from the terminal end. By the Wangensteen and Rice[29] method a marker is placed on the proctodeal site and the distance between this and the terminal gas shadow measured (Fig. 28). In rectal agenesis the distance is greater than 1·5 cm. and in anal anomalies less than 1·5 cm. We prefer Stephen's[10,17] method whereby the relation of the gas shadow to the pubococcygeal line (upper border of symphysis pubis to last piece of sacrum) is determined. In rectal malformations the bowel terminates on or above this line (Fig. 29), while in anal anomalies it stops well below the line (Fig. 30) at, or caudal to, the lowest ossified segment of the ischium.[10,17]

E. Treatment

As a general principle early operative treatment is needed in every case.[14] This is particularly important in cases with rectal agenesis or imperforate anus with acute obstruction, but in all cases delay carries with it the risk of excessive stretching of the rectum and later 'rectal inertia' with chronic constipation and overflow incontinence.

Rectal Agenesis

The treatment is difficult because a high urinary fistula usually has to be dealt with and the bowel brought through the pelvic floor in front of the puborectalis sling before an orifice can be fashioned. A simple perineal approach is inadequate and destructive and is now universally condemned.[10,14,17,22] A combined abdominoperineal pull-through operation such as described by Rhoads *et al.*[30] must

be performed; this is best done soon after birth. Infants of 3 days and less tolerate this extensive operation very well.[1] After this age, however, the operation becomes extremely difficult because of distension, and then a transverse colostomy should be performed. Colostomy is also necessary in premature infants weighing less than 4½ lb. and in infants with other serious malformations. In such cases the definitive operation should not be unduly postponed because of the risk of urinary infection. We usually wait until the child weighs 10-12 lb. (age 3-4 months).

Anal Anomalies

These anomalies can all be rectified by a relatively simple perineal procedure.[6,9,10,14,17]

1. *Ectopic anus.* In the perineal variety operation is not justified, but regular dilatations are required. Vestibular anus is treated by the Denis Browne 'cut-back' technique.[9] One blade of a pair of scissors is inserted into the anal opening and guided posteriorly under the skin by external palpation until it approaches the natural anal site. Then the scissors are closed, thus performing a midline episiotomy (Figs. 18a, b, c, d). The wound edges are not sutured and post-operative dilatations are carried out daily for 3 months and then over longer intervals for the next 3-6 months. Vaginal ectopic anus may be similarly treated, but the orifice tends to remain tilted into the posterior wall of the vagina and secondary transplantation of the anus to the normal site is almost always necessary. (The best time for this procedure is probably the 4th year.[14] Alternatively, the orifice may be dilated regularly until the infant is about 6 months old, when primary transplantation of the misplaced anus may be performed.[31])

2. *Covered anus.* This condition is treated by a somewhat similar 'cut-back' technique. The 'track' is laid open from the front backwards, up to the point where it dips down to reach the anal site (Fig. 19). Epithelial excrescences are trimmed off to avoid tags. Regular post-operative dilatations are also necessary. (In girls with 'anovulvar fistula' the cut-back procedure is particularly satisfactory.)

3. *Imperforate anus.* This is the only anomaly that is treated by a formal perineal exploration through a midline incision extending from the scrotum or vagina in front to the coccyx behind. Preliminary 'needling' of the perineum to determine the exact depth of the blind end may be useful. The plane of dissection must be immediately posterior to the bulbocavernosus and during mobilization the levator 'sling' must *not* be divided but retracted. Prolonged post-operative dilatations are necessary.

4. *Imperforate anal membrane.* This anomaly is easily treated by cruciate incision of the membrane followed by dilatations.

F. Prognosis

There is a great difference in the prognosis of rectal and anal malformations. In *rectal agenesis* the prognosis is poor for the following reasons:

1. The infant soon develops acute intestinal obstruction, and delay in diagnosis is often responsible for pre-operative or operative fatalities.

2. Associated anomalies which may be incompatible with normal life are common.

3. A major abdominoperineal operation, which in itself is a serious risk, offers the only hope of a reasonable result.

4. The results of surgical treatment are poor because:

(*a*) Post-operative strictures are common (Fig. 20).

(*b*) Continence is defective on account of lack of normal rectal sensation, absence of external anal muscles, and often poor development of the levator[10] (Fig. 21). However, these children can usually be trained to social cleanliness when old enough to cooperate, and this is preferred to a permanent colostomy. Despite most extravagant claims by many American surgeons, we are convinced that normal continence is impossible except in those rare cases that have a complete proctodeum. However, it is our impression that the earlier the definitive operation is done, the more satisfactory are the functional results.

In *anal anomalies* the prognosis is excellent provided ill-advised attempts at 'perineal mobilization' and 'transplantation of fistulae' have been avoided. Acute obstruction is rare. Other serious malformations are uncommon. The defects can usually be corrected by a simple procedure and continence is very satisfactory in most cases. Stricture formation with the development of chronic constipation and rectal inertia is, however, an important sequela which must be prevented by regular post-operative dilatations. Providing the enlargement of the anus has been adequate and is kept adequate, (Figs. 22-24) bowel function is virtually normal, and in females normal childbirth can take place (Fig. 25). This is indeed fortunate, because in our experience these 'lesser anomalies' occur in more than half of the cases of 'imperforate anus'.

CONCLUSION

In conclusion, I can do no better than quote from a recent article by John Scott,[19] who has done a great deal of work on this subject both at the Hospital for Sick Children, Great Ormond Street, London, with Harold Nixon, and at the Boston Floating Hospital for Children, with Orvar Swenson:

'The surgical treatment of the imperforate anus is difficult and fraught with many disappointments. Only those surgeons who have special training and experience in operating on these small patients and who are prepared to supervise the long period of bowel training which is usually required should undertake their management. Parents must be encouraged to persevere with the unpleasant tasks that are often required of them, and the surgeon must place himself at their disposal at all times to give advice and hope. Only special pediatric surgical centres can provide the expert and efficient nursing care which is so valuable in these cases, and with modern communications babies can be transported rapidly and safely. There is no greater tragedy than the child condemned to a lifetime of misery and embarrassment by errors in the initial surgical treatment and lack of interest in the after care'.

ADDENDUM

Since the completion of this article another 14 patients suffering from malformations of the anus and rectum have been treated. The distribution of the various types of lesion was in keeping with our previous experience, but, strangely enough, there were 2 cases of imperforate anal membrane. Both these babies presented with acute intestinal obstruction. In the first a colostomy was performed elsewhere, and in the second the presence of a membrane situated approximately 1 cm. from the anal verge and *not* bulging through the anus, was diagnosed on clinical and radiological examination, and therefore treated by a cruciate incision and subsequent dilatations.

I wish to thank Dr. J. G. Burger, Superintendent, Groote Schuur Hospital, Cape Town, and Dr. J. Mostert, Superintendent, Red Cross War Memorial Children's Hospital, Cape Town, for permission to publish the case analyses and photographs.

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IMMUNIZATION OF THE RURAL BANTU AGAINST DIPHTHERIA IN THE NORTHERN TRANSVAAL

I. IMMUNITY PRODUCED AFTER ONE, AND AFTER TWO, INJECTIONS OF ALUM-PRECIPITATED TOXOID

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MARY ROBINSON and J. H. MASON,* *South African Institute for Medical Research, Johannesburg*

In the rural Bantu population of the Northern Transvaal diphtheria has occurred sporadically during the last 10 years, with no major outbreak and with no obvious seasonal distribution. In Table I the incidence per 100,000 of the population (all *rural* Europeans and all *rural* Natives) is recorded. The difference in incidence between the races is striking.

TABLE I. RURAL DIPHTHERIA IN NORTHERN TRANSVAAL: AVERAGE ANNUAL INCIDENCE PER 100,000, 1951–57

District	*Rural European*	*Rural Bantu*
Barberton	68·8	7·5
Groblersdal	13·8	20·2
Letaba	58·9	4·0
Lydenburg	27·8	9·0
Nelspruit	34·8	9·5
Pietersburg	*21·0*	*6·4*
Pilgrims Rest	10·3	2·4
Potgietersrust	17·4	6·0
Sibasa	—†	0·4
Soutpansberg	24·5	5·8
Warmbaths	12·5	3·9
Waterberg	15·8	14·1

† No Europeans

	Population 1951 census	*Population 1957 estimate*
Rural European	54,470	60,401
Rural Bantu	1,071,560	1,430,310

The incidence in the Pietersburg district, where the experiment about to be recorded took place, is given in Table II. Although these incidences are unsatisfactory from a public-health standpoint, it is clear that no major outbreak took place. In 1950, when 38 cases, the highest incidence, were seen, the monthly distribution from January to December was as follows: 0, 10, 2, 9, 7, 5, 2, 0, 1, 0, 2, 0.

TABLE II. NUMBER OF CASES OF DIPHTHERIA IN THE RURAL BANTU POPULATION OF PIETERSBURG DISTRICT 1948 - 57

Year	1948	49	50	51	52	53	54	55	56	57	Total
No. of Cases	10	32	38	12	23	9	7	17	11	15	174

Rural Bantu Population: 1951, 184,102: 1957, 232,511

In Table III the age distribution of rural Bantu diphtheria is given, from 1951 to 1957 for all the districts of the area and from 1948 to 1957 for the Pietersburg district. The age distribution is similar in both areas, indicating that the same kind of conditions prevail generally and that the experimental results obtained in the Pietersburg district would be valid for the 11 other districts. The relatively low incidence in the 0–1 year group could have been predicted in view of the work of Mason *et al.*,[1] who showed that young Bantu babies, both rural and urban, have a high level of circulating antitoxin, maternally transmitted. When this decreases and eventually disappears after the age of about 6 months the incidence of diphtheria increases, to decline only after the age of 9 years, when silent and overt infections, spread over the years, have stimulated the production of a satisfactory active immunity.

TABLE III. PERCENTAGE AGE DISTRIBUTION OF RURAL BANTU DIPHTHERIA

Age Groups (years)	*12 Districts 1951–1957*	*Pietersburg District 1948–1957*
0—1	3·45	5·97
0—4	48·28	50·09
0—5	56·90	58·23
0—9	81·03	84·30
10—14	8·62	7·04
15—19	4·60	3·44
+19	5·75	5·24

In a large area like the Northern Transvaal, where the rural Bantu population is scattered, communications poor, immunization centres rather widely separated and the available trained medical personnel (medical officers, nurses and aides) already occupied with many other aspects of preventive

* Reprints obtainable from J. H. Mason, South African Institute for Medical Research, Johannesburg.

medicine, it is sometimes difficult to carry out a full diphtheria immunization programme, viz., the injection of 2 or 3 spaced doses of a prophylactic followed later by boosters.

It is unnecessary, in this article, to present evidence showing that the injection of 2 or 3 spaced doses of a diphtheria prophylactic produce a better immunity than 1 injection; this has been proved in many tests the world over. Further, it is almost profitless to review the literature dealing with the duration of immunity following immunization because as Bojlén and Scheibel[2] point out, the antigens used by different workers have not been the same and the number and spacing of the injections have varied. Further, and of great importance, the occurrence of silent infections plays a big part in maintaining and stimulating immunity in those who have received a basic immunization. It is obvious that the results of a particular experiment will have a bearing mainly on children living under the same conditions of housing, hygiene and climate, and with the same chance of exposure to inapparent infection, and need not necessarily be applicable to others living under different conditions.

Bearing in mind that diphtheria occurs only sporadically in the Northern Transvaal, that personnel is somewhat small in numbers, and that the assembling of children and of mothers with babies is difficult (a walk of from 10 or 15 miles is often involved), we decided to compare the immunizing effect of 1 and of 2 injections of alum-precipitated toxoid (APT). Even if the basic immunity produced by 1 injection was not so good as that produced by 2 injections, and this was to be expected, there was a chance that silent infection would maintain or increase this immunity more effectively in children so immunized than in those originally not immune and not immunized.

MATERIALS AND METHODS

Locus. Mphahlele's Location in the south-west portion of the Pietersburg district was chosen because it was reasonably accessible to one of us (H.H.E.), a regional health officer stationed at Pietersburg. It has a fairly stable, essentially rural Bantu population numbering between 12,000 and 15,000 and has few outside contacts.

Antigen. Alum-precipitated toxoid, 50 Lf per ml., was injected subcutaneously in a dose of 0·5 ml.

Selection and immunization. Blood samples were taken from 262 babies aged 6–24 months, sexes being equally divided. The sera were assayed for antitoxin by the intracutaneous method in guinea-pigs, using a stable toxin able to detect 0·001 unit. Only those children were selected for inclusion in the main experiment whose serum contained $<0·001$ unit of antitoxin per ml. They were divided into 3 comparable groups:

Group 1 received 2 injections of APT with an interval of 6–8 weeks between the injections.

Group 2 received 1 injection of APT.

Group 3 received no injection and served as controls.

Blood was taken from every child who presented himself, 3, 6, 12 and 24 months after the start of the trial, and the sera were assayed for antitoxin.

The sera from the original 262 children were initially titrated at 2 levels only—for 0·001 unit and 0·002 unit per ml.—and only those with $<0·001$ unit per ml. were included in the main experiment. Another group was formed from those whose serum contained $>0·001$ unit or $>0·002$ unit per ml., and constituted *group* 4. These received 1 injection of APT and were bled and their sera assayed 3 and 12 months later. In this way, a comparison could be made between their subsequent immunity and that of group 2 (no initial antitoxin, 1 injection of APT).

In addition, 3,825 children, including all those below the age of 12 years, received a 1st injection of APT and 3,084 of them a 2nd injection 8 weeks later, with the object in view of reducing the chance of an outbreak of diphtheria in the area or of the occurrence of mild undetected disease, both of which could lead to inapparent infections and antibody production in the groups under test. However, as concerns the elimination of silent infections, this end was not achieved.

RESULTS

Of the 262 sera initially assayed, 104 had $<0·001$ unit per ml., 108 had $>0·001$ or $>0·002$ unit per ml., and in 50 the result was doubtful, i.e. there was an indication that they contained some antitoxin but less than 0·001 unit per ml.

Most of the children were between the ages of 1 and 2 years, a few were between 6 and 12 months and the sexes were equally divided. The results, examined statistically, revealed no differences referable to age or sex and, for this reason, they were pooled.

The result of antitoxin assays carried out 3, 6, 12 and 24 months after the start of the experiment are given in Fig. 1. On the assumption that 0·004 unit per ml. of serum will produce the Schick-negative state and that 0·04 unit per ml. will confer immunity, the percentages referring to these states are included in the chart.

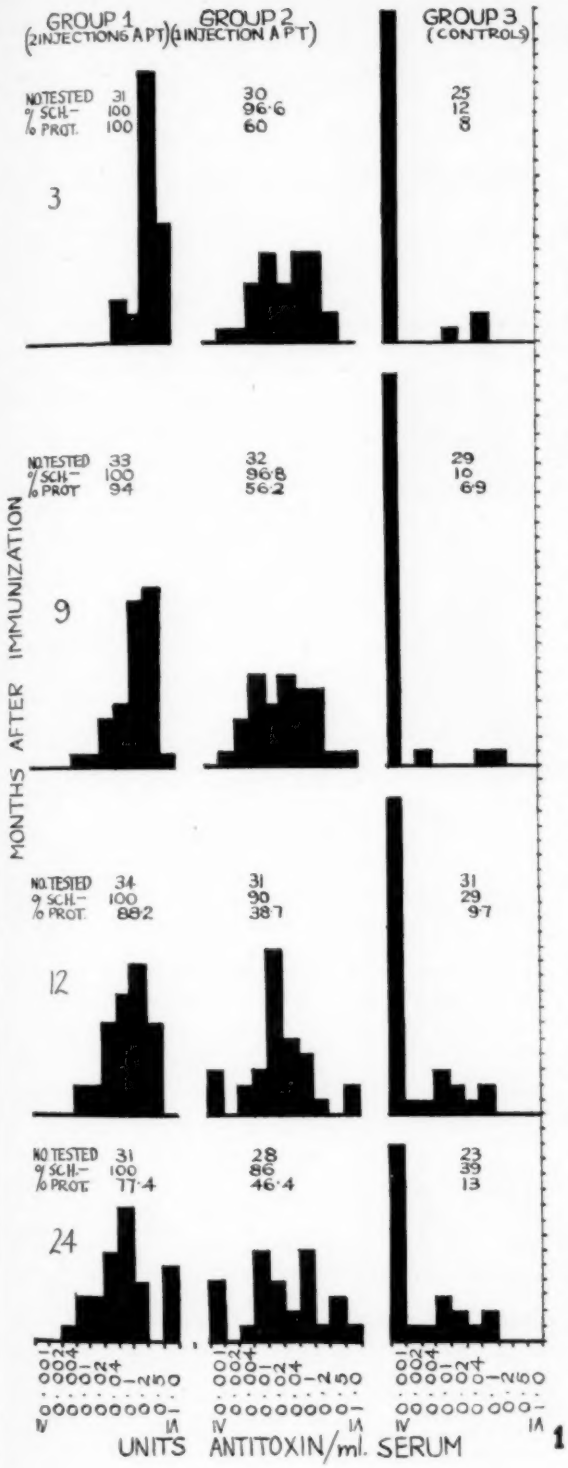
A consideration of the results given in Fig. 1 shows clearly that 2 injections of APT produce a significantly better immunity than one and this holds for all the 4 time intervals. Even after 2 years no child was Schick-positive in group 1 (2 injections), whereas 14% were Schick-positive in group 2 (1 injection). However, every child in group 2 had some circulating antitoxin after 3 and 6 months. Silent infection was obviously at work, shown clearly by the development of some immunity in some of the controls. It can be assumed that this type of stimulation was taking place in the majority of the children and certainly played a part in maintaining and boosting immunity throughout the course of the experiment.

Although, as expected, the 2-dose method produced the better immunity, the 1-dose method cannot be discarded as valueless. It was instrumental in laying down such a basic immunity that the majority of the children were Schick-negative for 2 years and that 60%, 56·2%, 38·7% and 46·4% were protected after 3, 6, 12 and 24 months respectively. This concerns only one group of children, selected because they had no demonstrable basic immunity. But when these results are pooled with those obtained from the children of group 4, who initially had some circulating antitoxin and who received 1 injection of APT, it is clear that a 1-dose method has a definite value in an area such as the Northern Transvaal.

In Fig. 2, histograms are given for the following groups and time intervals:

Group 1, originally not immune, 2 injections of APT, as in Fig. 1, 3 and 12 months.

Group 2, originally not immune, 1 injection of APT, as in Fig. 1, 3 and 12 months.



Group 4, originally immune, 1 injection of APT, 3 and 12 months.

Groups 2 and 4 pooled, 3 and 12 months.

A comparison of the results leads to the following conclusions:

1. *Group* 1 *versus Group* 2

These have been discussed when dealing with Fig. 1.

2. *Group* 2 *versus Group* 4

(*a*) *After* 3 *months*. The general level of immunity is much higher in group 4 than in group 2 although neither the percentages Schick-negative nor the percentages protected (96·6% and 60% for group 2 and 100% and 82% for group 4) differ significantly.

(*b*) *After* 12 *months*. Again the general level of immunity is higher in group 4 than in group 2. The percentages Schick-negative do not differ greatly (90% for group 2 and 100% for group 4) but the percentages protected differ significantly (38·7% for group 2 and 91% for group 4).

3. *Group* 1 *versus Group* 4

(*a*) *After* 3 *months*. All the children of each group are Schick-negative and 100% of group 1 and 82% of group 4 are protected, a statistically significant difference.

(*b*) *After* 12 *months* All children of each group are Schick-negative and 88·2% of group 1 and 91% of group 4 are protected, a difference which is not statistically significant. The general level of immunity is similar in each group.

4. *Group* 1 *versus Groups* 2 *and* 4 *pooled*

(*a*) *After* 3 *months*. The percentages Schick-negative are nearly the same, but significantly more are protected in group 1 (100%) than in groups 2 and 4 (72·4%).

(*b*) *After* 12 *months*. The percentages Schick-negative are nearly the same (100% for group 1 and 95·3% for groups 2 and 4) but the percentage protected is significantly higher in group 1 (88·2%) than in groups 2 and 4 (65·6%).

DISCUSSION

We should be loath to present this report if the results, or our interpretation of them, could be taken as advocating a retrograde step in the control of diphtheria by immunization, viz., the substitution of the proven 2- or 3-dose method by a single-injection procedure. As already stated, we carried out this experiment in an attempt to solve a problem concerning one special group of people. The question we set out to answer was: 'If a 2-injection campaign is so difficult to carry out as to be nearly impossible of attainment, should any immunization at all be attempted and, if so, would it be worth while?' We feel that we have answered both parts in the affirmative. About 50% of Bantu babies between the ages of 1 and 2 years have some small degree of immunity, produced by silent infections or by very mild attacks of diphtheria. In some of them at least, a single injection of a potent antigen acted as a secondary stimulus, if not as a booster. A single injection produced some immunity in all those without initial detectable circulating antitoxin, albeit low in a few and waning in some. But 1 year after this antigenic stimulus,

Fig. 1. Showing number of children, with antitoxin titres (units per ml. of serum) 3, 6, 12 and 24 months after 2, 1 or 0 injections of APT. Sch.— = Schick-negative. Prot. = protected. 1 square (i.e. 1 notch vertically) = 1 person.

among 64 children, originally with or without some degree of immunity, 61 (95·3%) were Schick-negative and 42 (65·6%) were protected. Among 31 control children, all initially without detectable immunity, 9 (29%) were Schick-negative and 3 of these (9·7%) were protected after 1 year.

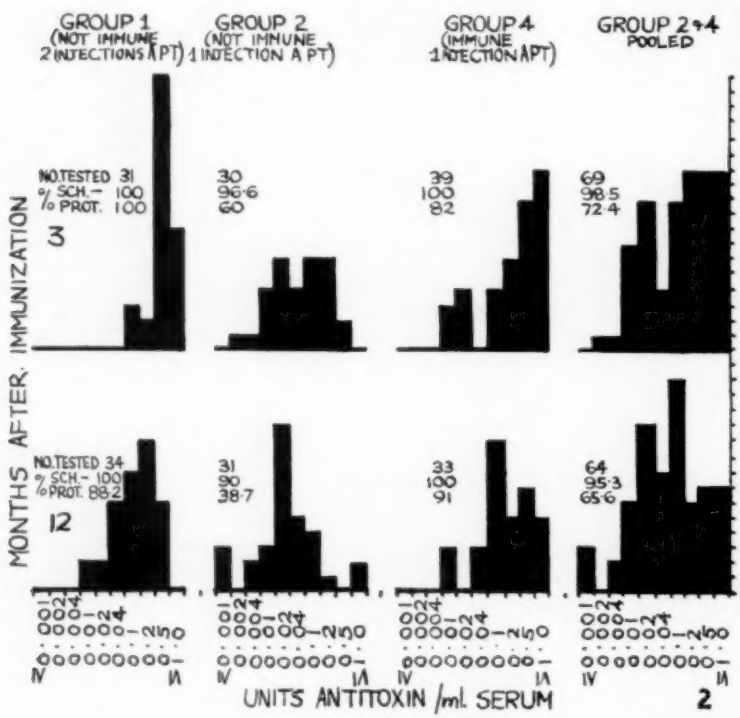


Fig. 2. Showing number of children in groups 1, 2, 4, and 2 plus 4, with antitoxin titres (units per ml. pf serum) 3 and 12 months after 1 or 2 injections of APT. Sch.—=Schick-negative. Prot.=protected. 1 square (i.e. 1 notch vertically)=1 person.

Thus, as silent infections only were responsible for producing immunity in the control children, it is obvious that such infections were at work in the immunized babies and were responsible, in some measure, for maintaining and boosting immunity in them.

In spite of the results obtained we must again stress that we do *not* advocate a 1-injection method for the control of diphtheria. But we are of the opinion that it has a place in an immunization campaign in areas *proved* to have conditions like those prevailing in the Northern Transvaal. We feel that it is better to confer *some* immunity on *all* the children of such a district rather than to omit immunization altogether because 2 injections cannot be given; or to immunize 10% of children with 2 injections and to leave the remaining 90% unprotected.

Diphtheria was not reported in Mphahlele's Location during the 2-year period embraced by the trial, but this does not rule out the possibility that mild attacks passed unnoticed. There is no means of knowing if immunization of the whole child population of the area reduced the number of carriers, but it is obvious that it did not eliminate them. The carrier problem is being investigated by one of us (H.H.E.) and will form part of the subject matter of another article.

CONCLUSIONS

1. Diphtheria occurs sporadically in the rural Bantu population of the Northern Transvaal. A major outbreak has not been recorded during the past 10 years.

2. Inapparent infections are common and are responsible for producing immunity in the non-immunized and for maintaining and boosting immunity in the immunized.

3. In about 50% of rural Bantu children between the ages of 1 and 2 years small amounts of circulating antitoxin are detectable.

4. Two spaced injections of alum-precipitated toxoid produce a better immunity than one injection.

5. Children with some degree of naturally produced immunity respond better to 1 injection of alum-precipitated toxoid than those without detectable natural immunity.

6. A 2-injection immunization procedure is advocated but a 1-injection method should be used rather than omit immunization altogether.

We have pleasure in thanking our colleague, Dr. P. Agerholm Christensen, for examining our results statistically.

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Dr. G. P. de Kock *by* Dr. A. W. S. Sichel.

Jannette Murray *by* Dr. A. W. S. Sichel.

Mev. A. P. Blignault *deur* Prof. F. D. du T. van Zyl, Drs. J. J. W. van Zyl, G. le Roux, A. D. Keet, E. G. van Hoogstraten en W. G. Davis.

Mrs. P. Eberlein *by* Drs. A. Amoils and H. Penn.

Mrs. B. Swil *by* Dr. Ruby Sharp and Dr. P. Jenkin.

Linda, daughter of Dr. A. P. Peires *by* Drs. A. Schiller and P. Jenkin.

Total received from Services Rendered: £194 13*s.* 0*d.*
Totaal ontvang vir Dienste Gelewer:

Donations : Skenkings:

Drs. M. Gelfand, B. Levin, B. Wainer; I. Wolpe, A. Beckitt, M. Fletcher, J. Opperman, A. Gordon; Dr. and Mrs. F. E. Meltzer; Drs. J. G. Berry; E. Bligh; S. Copans; M. G. Myers; W. A. van der Merwe; J. Q. Ochse; C. F. Scheepers; M. Taylor-Smith; M. B. Toerien; F. E. Mentz, H. M. Viljoen, C. E. M. Viljoen, le Roux, Morkel; Potash, Kramer, Levin, Stoch; King, Prinsloo, Botha; A. Gould; Nieuwoudt, Gilliland; M. Symon; B. Botha; Mynhardt, Snyman, Gough, Botha; Haine, Fowler and Steynberg; H. Feldman; W. M. Bezuidenhout; M. R. Mellett; D. M. Lithgow, Dr. R. E. P. Matthews, Dr. L. Fernley; A. F. Richey, Dr. A. J. P. Lessing, Dr. G. L. St. J. Whittington-Wriford; E. Krause; N. M. Shear, R. W. Williams, L. Shulman, J. L. Parker; L. H. Botha, F. L. Potter, M. E. Brown, and T. H. R. Bohlmann.

	£	s.	d.
Total : Totaal	56	14	0
Northern Transvaal Branch (M.A.S.A.)	2	2	0
Cape Western Branch (M.A.S.A.), Collection Box	8	14	4
Medical Wives Association of Port Elizabeth ..	11	0	0
Proceeds of functions by Medical Wives, Association of Port Elizabeth	30	0	0
Anonymous	10	0	0
Southern Transvaal Branch (M.A.S.A.), proceeds of merry-go-round function	2,532	7	0
Total Donations : Totale Skenkings	£2,650	17	4
Grand Total : Groottotaal	£2,869	13	4

IN DIE VERBYGAAN : PASSING EVENTS

Cape Western Branch (M.A.S.A.), Leipoldt Memorial Lecture. The second Annual Leipoldt Memorial Lecture will be delivered at an open meeting of the Branch on Friday 16 October at 8.15 p.m. in the New Science Lecture Theatre, University of Cape Town, Rondebosch, Cape. The speaker will be Dr. D. P. Marais, President of SANTA, and the subject of his lecture will be 'Whither tuberculosis'. Members and their wives or husbands and friends are welcome to attend this meeting. Refreshments will be served in the Students' Union after the meeting. The telephone number of the venue is 69-9932.

* * *

University of the Witwatersrand, Medical Graduates Association. A full-time, intensive, postgraduate course for general practitioners will be held from Monday 18 January to Friday 22 January 1960. The course will consist of practical demonstrations, ward rounds, and symposia. Senior members of the Hospital and University Departments will participate. The course will include paediatrics, anaesthetics, dermatology, and orthopaedics, in addition to general medicine, surgery, and obstetrics and gynaecology.

The fee for the course is £5, payable in advance to the Registrar of the University of the Witwatersrand. Assistance will be given in arranging accommodation for those desiring it. Only a limited number of practitioners can be accommodated. Applications should be made to the Medical Graduates Association, Medical School, Hospital Street, Johannesburg, not later than 25 November 1959.

* * *

Society of Clinical and Experimental Hypnosis. At the Annual General Meeting of this Society held in Johannesburg on 25 August, the following office bearers were elected: Dr. B. W. Levinson (Sterkfontein Hospital), President; Dr. S. Rootenberg, Vice-president; and Mr. A. L. Coetzee (Northlea Retreat), Hon. Secretary/Treasurer. Additional committee members elected were Drs. Y. le Jeune, C. Shubitz and E. Sack. This Society is at present seeking affiliation with the International Society. Applications for membership are invited from the medical profession, dentists, and psychologists who are registered with the South African Medical and Dental Council. Those interested should contact the Hon. Secretary at P.O. Box 20, Bergvlei, district Johannesburg.

* * *

Potchefstroom Division, Southern Transvaal Branch (M.A.S.A.). A clinical meeting will be held on Friday 30 October at 8 p.m. in the Klerksdorp Hospital. At this meeting Prof. James T. Louw, Professor of Obstetrics and Gynaecology, University of Cape Town, will speak on 'Views on the treatment of genital cancer' and Dr. W. H. Muller, Klerksdorp, will speak on 'Functional uterine bleeding'.

Afdeling Potchefstroom, Tak Suid-Transvaal (M.V.S.A.). 'n Kliniese vergadering sal gehou word op Vrydag 30 Oktober om 8 nm. in die Klerksdorp-Hospitaal. By hierdie geleentheid sal prof. James T. Louw, Professor van Obstetrie en Ginekologie, Universiteit van Kaapstad, 'n lesing gee oor 'Behandeling van genitale karsinoom', en dr. W. H. Muller, Klerksdorp, sal 'n lesing gee oor 'Funksionele bloeding van die uterus'.

* * *

Prix du Cinéma Médico-chirurgical. The Annual Prize for 'Medico-surgical Cinema', awarded by *La Presse Médicale*, which comprises 100.000 francs cash (with a possibility of being divided) and various other awards, will be given during the last session of the course of *Actualités Médico-chirurgicales* at the *Nouvelle Faculté de Médicine de Paris* on 5 April 1960. The jury will consider the didactic value of the film as well as its proper cinegraphic quality. Only 16 mm. films will be admitted. Entries and films are to be sent to the *Secrétariat du Journal 'La Presse Médicale'*, 120 Boulevard Saint-Germain, Paris VI°, before 29 February 1960. Awards will be remitted to the authors of the best film. Contrary to former years, films subsidized or produced by a laboratory or firm will not be excluded from the competition.

South African National Tuberculosis Association. The chief work of local SANTA branches throughout the country is the assistance of families and dependents of TB sufferers. This work is done, not only on humanitarian grounds, but because it plays an important part in preventing the spread of the disease. First of all when the family, especially the children, are well nourished there is far less likelihood of the contacts contracting TB, and secondly, if the TB sufferer himself knows his family will not be left destitute he is more likely to undergo treatment and not to continue moving about in the community spreading the disease. In order to obtain factual information regarding the expenditure on this 'care work' (as it is known in SANTA) and in order that representations based on definite figures may be made to the Government for some form of official subsidy, a comprehensive questionnaire was recently issued to all SANTA branches by the National Office.

The results are startling. They indicate that at present an amount of nearly £70,000 per annum (collected from the public) is spent on the provision of food, clothing, rents, medical expenses and rehabilitation for TB families. All branches indicate that their work is limited by the funds available and that if they were to provide adequately for all demands in their areas a figure of between £190,000 and £200,000 per annum would be necessary. No administrative or indirect expenditure is included in these estimates.

In addition to the approaches SANTA intends making for official financial assistance for this vital work, the Association has recently appointed a Branch Liaison Officer. He is at present undertaking a comprehensive investigation into all sources of official assistance available under existing legislation including disability grants for patients and maintenance grants for their families. His findings will be made available to all local TB Associations and it is intended that he should visit each one in turn meeting local branch and departmental officials and helping to unravel local problems in connection with the assistance of TB families.

* * *

Rehabilitation Centre. The establishment of a rehabilitation centre for mine Native paraplegics has been agreed to in principle by the medical committee of the Transvaal and Orange Free State Chamber of Mines. This fact emerges from the 1958 report of Dr. J. H. G. van Blommestein, Medical Consultant to the Anglo American Corporation of South Africa Ltd., who states, 'With the increasing desire today on everyone's part to do everything possible for the Native mineworker, it was considered that the time was opportune to make an active investigation into the question of the rehabilitation of Native paraplegics'. The matter had been under consideration since 1957 and, in discussions with the medical officers of other mining groups, it became evident that any such scheme could only be approached on an industry basis. A report dealing with various aspects of the matter was drawn up and submitted to the medical committee of the Chamber of Mines, which whole-heartedly supported the idea. It now remains for the best method of implementing the project to be ascertained.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Tuesday 20 October at 12 noon in the Bennie de Wet Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Dr. H. E. Schendel will speak on 'Changes in serum polyenoic acids during the treatment of kwashiorkor'. All who are interested are invited to attend this meeting.

BOEKBESPREKINGS : BOOK REVIEWS

CLINICAL NEUROLOGY

The Neurologic Examination. Incorporating the fundamentals of neuroanatomy and neurophysiology. 2nd edition. By Russell N. DeJong, M.D. Pp. xv+1078. 368 illustrations. $20.00. New York: Paul B. Hoeber, Inc. 1958.

The second edition of this valuable reference book follows 8 years after the first, and much new material has been added to cover the recent advances in many areas. The book is unique in that anatomy and physiology, physical examination and clinical diagnosis are treated concurrently. The extensive and excellent index adds to the book's usefulness as a source of neurological information. Current advances in neuro-ophthalmology, parietal-lobe function, the extrapyramidal motor system, and the brain stem reticular system, are dealt with. The meticulous description of methods of special investigation includes spectrophotometric tests of the cerebrospinal fluid. H.W.

BOECK SE SARKOÏDOSE

Der Lungenboeck im Röntgenbild. Von Prof. Dr. K. Wurm, Prof. Dr. H. Reindell und Prof. Dr. Dr. h.c. L. Heilmeyer. viii + 220 Seiten. 180 Abbildungen in 214 Einzeldarstellungen. DM 78.00. Stuttgart: Georg Thieme Verlag. 1958.

Die Franse dermatoloog Besnier het in 1889 sekere velveranderinge beskryf wat hy ‚lupus pernio' genoem het. Na 'n histologiese studie het die Noor, Boeck, in 1899 die toestand sarkoïed genoem, maar later die benaming verander na miliêre lupoïed.

Dit is vandag bekend dat Boeck se sarkoïdose (of beter, miskien, Besnier-Boeck se siekte) 'n gestelsaandoening is, maar die etiologie is nog net so onseker soos deur die verskillende benamings te kenne gegee word.

In die huidige monografie word beweer dat min nuwe kennis by ons kliniese begrip van die toestand gevoeg is oor die afgelope 40 jaar. Hoewel die name van 'n paar vroeëre ondersoekers in die teks genoem word, word hoegenaamd geen bibliografie verstrek nie. Dit is 'n leemte, veral as daarop gelet word dat 'n standaard leerboek meer as vyftig verwysings na vroeëre werkers in hierdie verband het. Die werk van Snapper, bv., word net terloops genoem. Dit laat die vraag ontstaan hoe volledig die oorsig oor die vroeëre arbeid is.

Tot sekere hoogte word hiervoor vergoed deur die groot hoeveelheid oorspronklike werk deur die skrywers self gedoen. Hulle het nie minder nie as 400 gevalle tot hulle beskikking gehad vir studiedoeleindes. Die meeste gevalle is ondersoek by 'n sentrale oogheelkundige kliniek, waarheen hulle verwys is vir aandoenings soos iridosiklitis en uveïtis.

Die longletsels in omtrent 60 afsonderlike gevalle word kortliks bespreek en geïllustreer deur middel van afdrukke van röntgenstrale van goeie gehalte. Die skrywers spreek hulle baie sterk daaroor uit dat die torakale letsels primêr in die mediastinale en brongiale limfkliere setel, sonder enige longaantasting (stadium 1). 'n Uitbreiding in die longe dui stadium 2 aan, en in stadium 3 vind daar bindweefsel-woekering plaas. 'n Spontane verbetering vind plaas in 80% van gevalle in die eerste twee stadia.

In 'n klein reeks gevalle is gevind dat die long- en klierletsels verbeter gedurende swangerskap. Daar is 'n goeie differensiële diagnose. Ten spyte van die leemte hierbo genoem, is daar baie stimulerende gedagtes oor die verloop van Boeck se sarkoïdose in die longe, en oor die röntgenologiese verskynsels. A.D.K.

MEDICAL HYPNOSIS

A Handbook of Medical Hypnosis. 2nd edition. By Gordon Ambrose, L.M.S.S.A. and George Newbold, M.B., B.S., M.R.C.S., L.M.M.S.A., D.R.C.O.G., D.C.H. Pp. xiii + 276. 27s. 6d. London: Baillière, Tindall and Cox Ltd. 1958.

With the increasing lively interest at present being shown in the clinical application of hypnosis, this handbook on the subject, in its clarity and balanced, reasonable approach, is a useful introduction for the practitioner or student. This second edition has been expanded to include new material and it will no doubt be as favourably received as its predecessor. A.H.T.

ANATOMY FOR SURGEONS : VOL. 3

Anatomy for Surgeons. Vol. 3. The Back and Limbs. By W. Henry Hollinshead, Ph.D. Pp. xii + 901. 785 illustrations. $23.50. New York: Paul B. Hoeber, Inc. 1958.

This volume deals with the anatomy of the back and upper and lower limbs. Each chapter is introduced by a detailed review of general considerations which is a valuable discussion of functional aspects of the appropriate anatomy. The individual chapters

are well written and cover the subject completely. The diagrams are excellent and consist of line drawings, many of them in colour. There are extensive references to the literature in the text, emphasizing that there is still controversy in detailed anatomy.

This is a most useful anatomical reference book, well written but unfortunately highly priced and, on the admission of the author, failing in the original intention of making it a portable reference.

A.E.K.

BIOCHEMISTRY OF THE CENTRAL NERVOUS SYSTEM

Biochemistry and the Central Nervous System. 2nd edition. By Henry McIlwain, Ph.D., D.Sc. Pp. vii+288. 45 illustrations. 45s. net. London: J. & A. Churchill Ltd. 1959.

The author of this excellent book has contributed much through his own research and teaching to the rapid development of biochemical studies of the central nervous system. He presents a reasoned exposition of the current status of the chemical aspects of the cerebral structures and of their origin, in regard both to individual cells and the brain as a whole. Biochemistry of the central nervous system overlaps and adjoins pharmacology (neuropharmacology and psychopharmacology), endocrinology, and other subjects. The various applications of chemistry to the study of the nervous system now receive the collective designation of neurochemistry, with some 6 groups and 40 sub-groups within the specialty; much of the work has appeared in journals of neurology and psychiatry. This book is essentially biochemical, but there is sufficient reference to neighbouring subjects to illustrate where they are applicable to biochemistry of the nervous system, and to the neighbouring subjects. Space limitation does not permit more than to state that this volume is a 'must' for students and research workers in medicine and science.

N.S.

A NEW SURGICAL TEXT-BOOK

Text-book of Surgery. Edited by Guy Blackburn, M.B.E., M.Chir., F.R.C.S. and Rex Lawrie, M.D., M.S., F.R.C.S., M.R.C.P. Pp. xii + 1122. Illustrations. 84s. Oxford: Blackwell Scientific Publications. 1958.

This new text-book of surgery written by the staff of Guy's Hospital is aimed at serving the needs of undergraduate students. The editors and authors of the various sections are men with a wide experience in the teaching of students, and they have attempted to cover in a comprehensive manner the whole field of general surgery. They have succeeded admirably and the student, already overburdened by the ever-increasing complexities and scope of the various subjects with which he has to contend, will find the book a valuable ally in his battle with the examiners. Certainly some chapters are better than others, a situation which is inevitable in a multi-author book, but the general standard is extremely high. A very welcome feature is the sections dealing with radiology and radiotherapy, and a very practical account of the mysteries of water and electrolyte requirements in surgical patients. In the mind of this reviewer there is no doubt that the book will run through many editions. The editors are to be congratulated on the birth of this new contribution from Guy's Hospital.

B.S.

THE DEAF CHILD

Hearing Therapy for Children. 2nd revised edition. By Alice Streng, M.A., Waring J. Fitch, M.A., LeRoy D. Hedgecock, Ph.D., James W. Phillips, M.D. and James A. Carrell, Ph.D. Pp. 353. Illustrations. $6.75. New York and London: Grune & Stratton, Inc. 1958.

This book should be of immense value to doctors, teachers and parents of children with hearing impairments. It stresses the teamwork necessary for the rehabilitation of these pupils, giving an outline and bird's-eye view of the medical aspect and treatment of deafness, the educational programmes advisable for children with varying degrees of hearing loss, and their social development.

Of particular interest are the sections on audiometry, stressing the importance of speech as well as pure tone audiometry and the emphasis placed on using every scrap of hearing. Excellent advice is given on the training of residual hearing. This is a book which contains very valuable advice on the needs of the hard-of-hearing and deaf child.

B.K.W.

PAEDIATRIC EMERGENCIES

Kinderärzliche Notfallfibel 4., ergänzte Auflage. Abwehr akuter Lebensbedrohung. Von Prof. Dr. B. de Rudder. viii + 205 Seiten. 34 Abbildungen. Ganzleinen DM 18.60. Stuttgart: Georg Thieme Verlag. 1958.

The author gives the essential features of diagnosis, differential diagnosis and emergency treatment of every disease in childhood which usually does or can present itself as an emergency. This calls for the utmost brevity in style, which makes reading difficult. To be thoroughly familiar with the booklet before any emergency arises seems therefore essential. So as not to waste valuable and often essential time in differential diagnosis, it is whenever possible set out for a whole group; for instance in meningitis the question is discussed how far the diagnosis has to be pushed before a basic treatment common to all forms of meningitis can be introduced. Advice on treatment is limited to the actual state of emergency, but the possibilities and requirements of a hospital in dealing successfully with these cases are outlined. Technical procedures required in emergency treatment are clearly and simply explained and an alphabetical list of all medicines mentioned in the text is given, with the appropriate dosage for the different ages. Since, however, most of the specialities mentioned in the book are not obtainable in this country this is of little help. Well reproduced photographs illustrate typical features of a disease where possible.

W.G.H.

HAEMATOLOGICAL TECHNIQUE

Haematological Technique. 2nd edition. By E. M. Darmady, M.A., M.D. (Camb.), F.R.C.P. and S. G. T. Davenport, F.I.M.L.T. Pp. viii + 244. 4 coloured plates and 23 text figures. 24s. net. London: J. & A. Churchill Ltd. 1958.

The second edition of this book serves as a most useful addition to other books on this subject, and covers both the theoretical and technical sides of haematology. Besides its undoubted value to medical technologists it should be of use to both medical students and trainee pathologists.

As the authors state, this book is primarily intended for medical laboratory technologists preparing for examinations, and as such its scope and content is naturally limited as compared with other books on haematology. However, with the introduction of chapters on blood grouping and serology, and the inclusion of a number of relatively new techniques which are now generally accepted in the investigation of certain blood disorders, this book well deserves a place in any routine haematology laboratory. S.J.R.

A NEW GYNAECOLOGICAL QUARTERLY

Clinical Obstetrics and Gynecology. Vol. 1. No. 2. June 1958. *Toxemias of Pregnancy.* Edited by Louis M. Hellman, M.D. *Fibromyomas of the Uterus.* Edited by Robert A. Kimbrough, M.D. No. 3. September 1958. *Symposium on Special Diagnostic Aids.* Edited by C. Paul Hodgkinson, M.D. *Symposium on Abnormal Uterine Bleeding.* Edited by John I. Brewer, M.D. No. 4. December 1958. *Symposium on Operative Obstetrics.* Edited by J. Robert Willson, M.D. *Symposium on Genital Cancer.* Edited by Daniel G. Morton, M.D. Published 4 times a year, over 1,100 pages. Illustrations. Sold by subscription only, $18.00 for 4 consecutive numbers. New York: P. B. Hoeber, Inc. 1958.

I was very disappointed in the first number of *Clinical Obstetrics and Gynaecology*, but in numbers 2, 3 and 4 there has been a remarkable improvement in the contents as well as the arrangement. If this standard is set in the forthcoming numbers, the series is well recommended to the busy gynaecologist who wishes to keep up to date and whose reading time is limited.

In the second number the physiological aspects as well as the clinical application of fluid metabolism and electrolyte balance is discussed in a simplified manner and to those of us who are not physiologists, but clinicians, it is of particular value. The authors have tried to give a full account of most of the well-known aetiological factors of toxaemia of pregnancy, but have

remained objective in their views on aetiology. Again we realize that we are no nearer to the solution than many years ago, but valuable suggestions are given on treatment. The symposium on fibromyomata of the uterus makes interesting reading in spite of the fact that most of it is text-book knowledge. The prominence lent to vaginal hysterectomies for fibromyomata of the uterus is however very dangerous, for it may persuade the inexperienced gynaecologist to attempt vaginal hysterectomies for fibromyomata which should have been treated by the abdominal route. We all know how difficult it may be to judge the size of fibromyomata, especially in the obese woman, and also how often other tumours are misdiagnosed as fibromyomata.

In number 3 the symposium on special diagnostic aids is to be commended; in no other book are all these aids discussed in such a compact manner. Although many of them, such as peritoneal washings and gynograms, are not practised in this country, it is well to know about them so that research on these lines may be carried on in our teaching hospitals. Abnormal uterine bleeding is discussed in detail and many aspects often forgotten by the practising gynaecologist are stressed. Functional uterine bleeding, which has always been a headache to most gynaecologists, is discussed in detail that is well worth reading.

Number 4, the symposium on operative obstetrics, can be recommended for the student studying for higher examinations. Many practical points which are often overlooked are discussed. It is however a pity that trial forceps are coupled with failed forceps under the same heading and that the advantages and importance of trial forceps are not discussed in more detail. The symposium on genital cancer is the best in this series so far and is discussed in an extremely readable and unbiased manner. The authors have stressed the aids to early diagnosis and also the importance of early diagnosis. What is very encouraging is the discussion of radiotherapy and surgery as adjuncts to teach other and not as the one versus the other as in most writings.

R.W.A.N.

FERTILITY

Studies on Fertility, Including Papers Read at the Conference of the Society for the Study of Fertility, Exeter, 1957. Being Volume 9 of the Proceedings of the Society. Edited by R. G. Harrison, M.A., D.M. Pp. x + 169. Illustrations. 25s. net. Oxford: Blackwell Scientific Publications. 1958.

This collection of papers on fertility include studies in the human male and female as well as the results of animal experiments in both sexes and biochemical assays of spermicidal agents.

A further interesting report on the questionable therapeutic effect of tubal patency tests is presented from Soho and University College Hospitals, where research in the matter is continuing. It would appear that pregnancy is just as likely to follow salpingography, insufflation, endometrial biopsy, or the mere passage of a uterine sound.

Amongst the animal experiments a study of the relationship between obesity and sterility in mice, and another on the mechanical influence of the vasa efferentia on the transport of radiopaque medium through the rat epididymis, are worthy of special mention.

J.P.R.

BRIEWERUBRIEK : CORRESPONDENCE

SOCIETY OF CARDIOLOGICAL TECHNICIANS

To the Editor: During the past few years cardiac surgery and cardiology have advanced in South Africa at an enormous pace. New diagnostic techniques and surgical procedures that were comparatively unknown 5 years ago are now common events in the larger cardiac centres, and with this rapid advance the technicians employed at these centres have likewise attained more importance, having to learn new methods and adapt themselves to new ideas (often with great difficulty, there being no recognized training scheme).

Unlike the other types of hospital technicians, the cardiac technician has no authoritative organization to promote a standard of training, nor is he able to qualify by examination. Both these factors severely limit his capabilities and those of the physician or surgeon by whom he is employed.

In a country so vast and so sparsely populated as ours, it is naturally difficult to incorporate such an organization without some assistance. It was with this view in mind that the Society of Cardiological Technicians of Great Britain was approached and a branch of that Society has recently been formed in the Union of South Africa.

The aims of the Society are:

1. To promote training in technical cardiology.
2. To maintain a recognized standard of training by means of examination.
3. To keep a register of those qualified.
4. Ultimately to obtain official recognition of its members.

The South African Cardiac Society has agreed that such a scheme of training would be desirable and approve its formation.

It is requested that those interested should write to the undersigned:

L. W. Piller
Chief Technician

Cardiac Clinic
Groote Schuur Hospital
Observatory, Cape
1 October 1959

HAEMORRHAGIC DISCHARGE FROM NIPPLE

To the Editor: I saw a woman, aged 58 years, who complained of an intermittent, bloody discharge from the right nipple of 3-week's duration.

On examination no tumour could be detected, but massage (pressure) of a strictly localized area about 1 inch above the nipple, caused a bloody discharge.

I referred her to a surgeon who informed me that surgery was indicated only when a tumour could be felt.

Now, according to Professor Saint, if a bloody discharge is produced by pressure over a localized area, whether a tumour is felt or not, surgery is indicated even if only for exploratory purposes. Christopher says that in the absence of a tumour mastectomy is indicated. Anson and Maddock say that a bloody discharge from the nipple is almost pathognomonic of a duct papilloma, which is definitely a precancerous lesion, and that such a case must be fully investigated.

I shall be glad to know what the consensus of opinion is regarding this matter.

C. Frank

Fraserburg, Cape
24 September 1959

DIE MEDIESE KONGRES EN AFRIKAANS

Aan die Redakteur: Ek het so pas teruggekeer van die Mediese Kongres op Oos-Londen. Dit was die eerste keer dat ek so 'n Kongres kon bywoon. Die Kongres was baie interessant en leersaam. Wat my en andere egter seergemaak het, was dat die President in sy Presidentsrede nie eenkeer Afrikaans gepraat het nie. As hy nie Afrikaans magtig was nie, kon ons hom nog verskoon, maar hy het die Administrateur in baie goeie Afrikaans verwelkom. Terloops, die Administrateur het vir hom 'n goeie voorbeeld in dié verband gestel.

Nog iets. Onder die lys van geneeshere aan wie medaljes toegeken is, was die naam van dr. J. N. W. Loubser (Posthuum). Elke lid se naam is afgelees en dan is 'n kort lewensskets van hom gelees. Wyle dr. Loubser se naam is nie eers genoem nie. Hoeveel mooier sou dit nie gewees het nie as een van sy kinders of 'n kollega-vriend sy medalje in ontvangs kon geneem het nie.

Plattelandse Geneesheer

2 Oktober 1959

[Dr. Loubser is voor sy dood deur die Sekretaris van die Mediese Vereniging uitgenooi om die medalje in ontvangs te kom neem. Omrede van sy swak gesondheid kon dr. Loubser nie die uitnodiging aanneem nie. *Soos gewoonlik* in sulke gevalle is reëlings toe getref om die motivering en medalje aan hom aan sy huis te oorhandig. Ongelukkig is hy voor dit kon plaasvind oorlede. Die medalje en motivering is tans by die Tak O.V.S. en Basoetoland. Die Kongreskomitee van Oos-Londen het per abuis sy naam op die program gelaat.—*Redakteur.*]